Canadian Journal of Surgery
Journal canadien de chirurgie

VOLUME 4 OCTOBER 1960–OCTOBER 1961

Chairman, Editorial Board: DR. R. M. JANES
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published by The Canadian Medical Association
150 St. George Street, Toronto, Ont.

VOLUME 4

No. 1	October 1960	Pages 1 - 144
No. 2	January 1961	145 - 262
No. 3	April 1961	263 - 404
No. 4	July 1961	405 - 502
No. 5	October 1961	503 - 610

HISTORY OF CANADIAN SURGERY

THOMAS GEORGE RODDICK

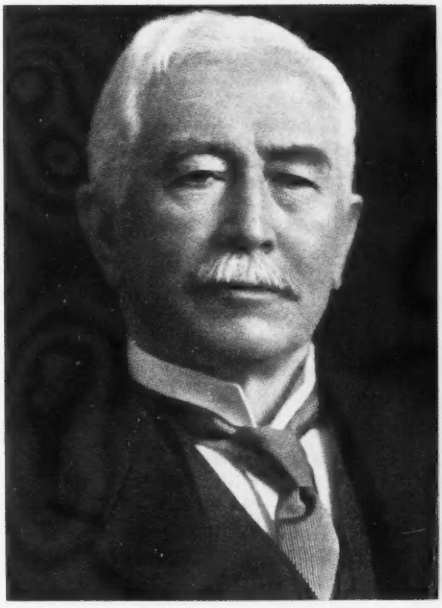
1846-1923

H. E. MacDERMOT, M.D., *Montreal*

THE NAME of Thomas Roddick is linked with the early use in Canada of Lister's antiseptic method more definitely than is the case with any of his contemporaries; there is no doubt that he hastened the acceptance of its principle. By his personality and character he also exercised a wide influence in his time, professionally, socially, and politically.

Born in Newfoundland in 1846, Thomas George Roddick began life with all the advantages of a good education. Like many of his contemporaries he intended to take his medical training in Edinburgh, but was persuaded to enrol at McGill University, where he graduated in 1868 with high honours. His energy and unquestioned ability made him a natural leader, and he rose to high teaching and administrative responsibilities as Professor of Surgery and later Dean of the Faculty of Medicine at McGill. He was one of the group in Montreal which included such men as Osler, Shepherd, Blackader, Buller and James Ross, who met frequently to dine as a club, and incidentally to develop the activity which is so well reflected in the medical journalism of the day. He soon began to contribute to the *Canadian Medical & Surgical Journal*, under the influence of its editor George Fenwick, who had been mainly instrumental in having him train at McGill.

Roddick graduated about three years after Lister had begun his work on antisepsis. It would be interesting to know what reference to Lister's work had been made in the surgery lectures at McGill at the time that prompted the question on Roddick's final surgical paper, "What is meant by the antiseptic treatment in surgery? In what cases is the use of carbolic acid especially indicated?" Certainly no one in Canada seems then to have grasped Lister's idea, although A. E. Malloch of Hamilton, Ontario, one of Lister's house surgeons in 1868, had begun using Lister's method in 1869.



Dr. Thomas George Roddick

In 1872 Roddick visited Edinburgh and saw Lister at work, but not until after a second visit in 1877 did he bring back Lister's carbolic spray and begin using it in his hospital work. Within twelve months he read a paper at the annual meeting of the Canadian Medical Association, describing his experience with antisepsis, and one year later he published the first comprehensive Canadian report on a series of cases thus treated.* These were from the wards of the Montreal General Hospital. He said that he had no "accurate comparative statement of the surgical results of the Hospital before and after the introduction of Lister's method", but his mortality rate was so strikingly low that his report carried immediate though perhaps not universal conviction. His comment was:

"We have then a record of 64 major operations with two deaths, being a mortality of

**Montreal General Hospital Reports*, Vol. 1, 1880.

3.12 per cent. There are few British hospitals whose statistics can compare favourably with these. The late lamented Callender of St. Bartholomew's boasted of a mortality of barely 3.4 per cent, his operations being performed by a modified antiseptic method.

"To give some further idea of the great change wrought by 'Listerism' in our own hospital, I would quote the results of the amputations of the lower extremity during the last two years of the old system. Of four amputations of the thigh recorded, *all were fatal.* Of six amputations of the leg, *four* were fatal."

It should be added that others in Canada, notably F. J. Shepherd, had shown an equally keen perception of the principles of Lister's work. Indeed, Roddick had been apt to cling too closely to the soon outmoded carbolic spray. But to him must be given the credit for an impetus in the adoption of antisepsis in surgery which no other surgeon in Canada could claim.

In 1885 there was a brief but extremely active interlude in Roddick's practice. The Riel Rebellion broke out in Saskatchewan in that year, and a force of about 5000 men was assembled to deal with it. Roddick was chosen as chief of the medical staff in the field, on the grounds that he was "one of the most distinguished Canadian surgeons, young, full of vigour, of powerful physique, knowing no fatigue, and a first class horseman." Of even more importance was his shrewd commonsense and natural administrative powers. All these qualities were now to be fully tested.

The campaign was short — March to August — but it presented peculiar difficulties in organization and transport, to say nothing of unusually harsh winter conditions at first. Roddick met all these with vigour and good judgment, and reports on the medical aspects of the campaign have nothing but praise for his work. It was the occasion for use of what was probably the first hospital ship in Canadian military operations. This was a special barge put into service by Roddick to transport the wounded from the field hospital at Saskatoon down the Saskatchewan River and Lake Winnipeg, to Winnipeg, a distance of 1100 miles, the trip taking 11 days.

There was remarkable freedom fro sickness amongst the troops. This wa ascribed vaguely to "the tonic and strengt ening properties of the northwest country" though these were of a somewhat heroi quality in view of the bitter weather whic the men, most of them raw recruits, ha to face without adequate protection du ing the first few weeks. It was fortunat that by the time warm weather arrive with the attendant possibility of typhoi fever, most of the fighting was over. Rod dick later spoke of the extraordinary free dom from infection amongst the operativ cases, not with any special reference t antisepsis, for he had little chance o operating himself, but realizing that th tent hospitals had not the background o old septic conditions.

For about the next ten years Roddick was fully absorbed with his teaching and professional work, as well as general medical affairs. In 1897 he was chosen president of the British Medical Association on the occasion of its first visit to Canada, in conjunction with the Canadian Medical Association — the first Canadian to be chosen for this position. Lord Lister was his guest on his visit to Montreal at that time.

The Canadian Medical Association has received from Dr. T. C. Routley an interesting historical link in the shape of the badge which was worn by Lord Lister at this combined meeting; it was pinned on to his lapel by Roddick. A friend of the Lister family gave it to Dr. Routley in England, when he was completing the same duties as President of the British Medical Association which Roddick had carried out 59 years before in Montreal. It was thought appropriate by the donor that the badge should come back to Canada through the medium of one who had himself occupied this combined office.

In 1894 Roddick began active work on what was to be his most notable achievement, that of establishing uniformity of medical registration throughout Canada. Ever since its formation in 1867 the C.M.A. had given close and constant attention to this subject. In spite of most devoted labours it was eventually admitted that the matter presented apparently insuperable

difficulties. At the root of these lay the fact of Provincial autonomy in matters of education — *all* education. Registration of doctors involved their education, and so far it had never been possible to persuade all nine sternly independent (and not overenthusiastic) Provinces to accept one uniform method.

Roddick was given the task of chairmanship of a C.M.A. committee on registration, and he began then his long uphill task of coordinating the views of the profession and soothing the sensibilities of nine Provincial licensing bodies. He actually managed to obtain most encouraging support from the profession on a report suggesting a form of Federal legislation. But as Parliament could not infringe on Provincial rights, and the Provincial legislatures would not unite to recognize a central board, an impasse developed.

Roddick was elected to Parliament in 1896, but as his party (Conservative) was then in opposition he could do little in the House and spent the next five years in influencing medical opinion throughout the country.

At last in 1901 he was able to bring in his Dominion Medical Bill. The essential feature of this was the formation of a Dominion Medical Council, whose diploma gave the right to practise throughout the Dominion. In 1902 he managed to get the Bill accepted in the House, and then came nine years of incessant labour to gain consent from each province separately, which had been conditional in acceptance of the Bill.

In 1911 his magnificent patience was rewarded by the passage of the Bill creating a Dominion Medical Council. Even Sir Charles Tupper, a master politician, had thought it was a hopeless venture and told Roddick that he would never be able to carry it through. Roddick was not essentially a politician. This particular achievement demanded qualities of tenacity, patience, good nature, and a knowledge of men, all of which may be found in a politician, it is true, but not necessarily with a personality such as his. His work was recognized by a knighthood in 1914.

Roddick is deservedly looked up to as a leading figure in Canadian medicine. He died in 1923, of a form of pernicious anæmia, with subacute combined sclerosis.

WILLIAM HARVEY IN RETROSPECT*

"The theme of this address has been suggested by the successful operation in Auckland over the past 12 months of the heart-lung machine. Inevitably our minds have been drawn the more closely to that great man who started it all, William Harvey. How he would have rejoiced in this working exemplification of his *de Motu Cordis et Sanguinis*. We might easily suppose that he could have foreseen, and perhaps did foresee this modern construction, in which 'he being dead yet speaketh'. It seems appropriate that we should again turn our minds to Harvey and his work, to see in retrospect their place in medicine historically and philosophically. . . .

"We need not look on the history of medicine as something divided from us by a great gap. History is what has happened right up to this 'now' when time-past meets time-to-come. Our own teachers in the faith are as significant as those whose names we remember only because they are attached to a structure, a symptom, or a disease complex. Today in the face of so great an accumulation of detail in physiology, pathology and therapeutics, the past seems to have been blotted out by the overwhelming present. . . .

"We can better estimate the greatness of Harvey when we remember that he worked and wrote without advantage of any magnification other than that of a hand lens and without knowledge of oxygen and oxygenation. His lecture notes show that as Lumleian Lecturer on Anatomy to the Royal College of Physicians he had been teaching the circulation of the blood for at least 12 years before he ventured into print. He was not himself unaware of the importance of his work . . ."

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*Robertson, H. G.: *New Zealand M. J.*, 59: 28, 1960.

ORIGINAL ARTICLES

SPONTANEOUS INTRACRANIAL HÆMORRHAGE: "SUBARACHNOID HÆMORRHAGE": A REVIEW OF INVESTIGATION AND TREATMENT IN 189 CASES*

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AN UNKIND FATE has so governed the chances of survival following spontaneous intracranial bleeding ("subarachnoid hæmorrhage"), that surgical intervention has become an urgent consideration in a large proportion of these cases. Unforgettable are those patients who recover from an initial hæmorrhage, only to succumb suddenly to unexpected recurrent bleeding a few days or weeks later. Furthermore, an operation becomes mandatory to preserve life and cerebral function in the large number of cases harbouring a discrete intracerebral clot (26% in this series).

The prevention of recurrent bleeding has become a problem of major surgical importance. This is particularly true of ruptured intracranial aneurysm, the chief offender, which has also proved the most difficult to eradicate safely. Although obliteration of the aneurysm is usually possible, the exposure with deep retraction and concomitant arterial spasm may precipitate a disastrous train of events leading to massive cerebral softening and swelling, with profound alteration of cerebral function or death. Surgeons have accepted the burden of proof that surgical treatment leads to a lower mortality rate than the 50% widely accepted under conservative regimes. With the knowledge that most aneurysms are small and accessible the pursuit of safer surgical methods will continue so that it may become possible to eliminate routinely the dread of recurrent bleeding.

Careful scrutiny of individual series of cases will serve to increase our knowledge of this catastrophe and further the development of effective treatment. This report is based on 189 patients who, before angiography, were considered to have bled from an intracranial arterial aneurysm or angioma. Patients with hypertensive cerebrovascular disease and those not having angiography have been excluded. Sixteen angiomas and 68 aneurysms were treated by direct surgical management. Seventy percent of the aneurysms were operated upon within one week of the last hæmorrhage and 50% were treated within 48 hours.

RESULTS OF INVESTIGATION

The presence of intracranial bleeding was verified in each case by lumbar puncture. Visualization of the cerebral arterial tree by the minimum of bilateral carotid angiography demonstrated in the majority of instances the nature and site of the offending lesion and the dynamics of the local and collateral circulation; without this information no rational plan of management or estimate of the prognosis can be made. Because of the danger of early recurrent bleeding, the general policy has been to perform bilateral carotid angiography shortly after admission, using appropriate compression to demonstrate the cross circulation. Although cerebral angiography is said to be not without risk at this stage, only one patient (who was seriously ill) suffered a fatal recurrence during the procedure and there were no other serious sequelae. The majority received premedication with papavarine and local anæsthesia.

Carotid angiography in 189 patients revealed the source of bleeding in 127 (Table I).

Vertebral angiography in 30 patients with negative carotid angiography demonstrated a lesion in a further eight cases. Thus angiography revealed the nature and site of the hæmorrhage in 135 of 189 cases or 71% (Table II).

PATIENTS WITH NEGATIVE ANGIOGRAPHY

Fifty-five patients, of whom 22 had vertebral angiography, appeared to have no

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*Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, January 1960.

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TABLE I.—Angiography in 189 Patients

Carotid positive	127
Vertebral positive	8 of 30
Total	135 or 71%

abnormality of the cerebral vascular system. Twelve have died of recurrent bleeding (Table III). This mortality of 22% was much higher than the 5% to 10% mortality generally ascribed to this group. Of the five autopsies performed, an aneurysm was found in four: carotid-posterior communicating, carotid bifurcation, middle cerebral trifurcation, and one on the right vertebral artery which ruptured and killed the patient on the morning of the proposed vertebral angiography. The survivors have been followed-up for periods varying from nine months to seven years, the average being 3.4 years.

A review of the arteriograms performed on the four patients who died from aneurysmal rupture did not disclose the lesion, but local spasm of the carotid artery was seen adjacent to the carotid aneurysm at the posterior communicating artery. On two occasions an aneurysm was demonstrated by repeat angiography. One was seen at the carotid-posterior communicating junction three weeks after the initial arteriogram which showed only intense local spasm. The second was at the basilar termination, 10 weeks and two hæmorrhages after initial filling of vessels of normal calibre. It is therefore important that, following initial negative angiography, the studies be repeated in two or three weeks or at least before discharge. Four patients had an exploratory craniotomy because the angiograms showed local arterial spasm or the questionable filling of a stump but in no instance was the source of bleeding discovered.

Patients With Tumours

Three patients bled from a tumour,

TABLE II.—Angiography in 189 Patients

	Cases	*Percentage*
Aneurysm	110	59
Angioma	21	11
Tumours	3	1
Negative	55	29
Total	189	

TABLE III.—Negative Angiography

Patients	*Alive*	*Dead*
55 (22 had vertebral angiography)	43 (78%)	12 (22%) (4 aneurysms found at post-mortem)

previously unsuspected: one with a middle sphenoid ridge meningioma, one a metastatic melanoma and one a metastatic carcinoma from the prostate.

Conservative Treatment of Patients With Verified Aneurysms

Forty patients, known to have bled from an aneurysm were not operated upon for a variety of reasons including age, associated disease, critical condition, technical difficulty or inoperability, or unwillingness on the part of the patient, attending physician or the family to accept the risk of operation. Three patients died with recurrent bleeding before a planned operation could be carried out. Spontaneous thrombosis apparently occurred in two aneurysms as shown by serial angiography. Because of this selection the group cannot be used for comparison with surgically treated cases (Table IV).

Arteriovenous Malformations

Twenty-one, or 11% of the patients proved to be bleeding from an arteriovenous malformation. In 19 the malformation filled through the carotid circulation but one pontine and one occipital angioma were visualized only by vertebral angiography. Intracerebral clots were found in 11 cases, principally with the smaller, more deeply seated malformations (Table V). Three angiomas were situated deeply within the hemisphere or in the midline and were considered inoperable and these

TABLE IV.—Verified Aneurysms—Conservative Management

A selected group—cannot be used for comparison

Grade	*No.*	*Alive*	*Dead*	*Disabled*
1	18	13	5	3
2	2	1	1	
3	5	4	1	2
4	2	1	1	1
5	13		13	
40 patients		19 alive (6 disabled)		
		21 dead (52.5% mortality)		

TABLE V.—Angiomas—21 Patients

2	Moribund (clots)—died
3	Central (no operation)—alive
16	(9 with clots)—intracranial operation —No operative mortality
	13—total removal
	2—major feeder clipped (2 late deaths—4 and 5 years)
	1—muscle pack

patients have remained well for two, three and five years respectively. Two patients with intracerebral bleeding were moribund with fixed pupils and died shortly after admission.

Sixteen patients, nine of whom were stuporous and hemiparetic with intracerebral clot, were operated upon with no mortality or serious morbidity. Artificial hypothermia has not been used in these cases. Total removal of the malformation and associated clot, if present, was accomplished in 13 cases. One small deep temporal angioma invading the side of the midbrain was superficially packed with hammered muscle and there has been no recurrent bleeding in the year elapsed. In follow-up, the results in all but one case with a preoperative hemiplegia were classed as "excellent" or "good". In two deeply seated midline lesions the hypertrophied major feeding arterial channel was clipped without incident and the postoperative angiograms showed absence of filling in one and greatly reduced circulation through the other. However, both these patients suffered massive recurrent bleeding five and four years later. The first died in a few hours as the result of the hæmorrhage and the second died following a craniotomy in another centre. Although there was no alternative in these cases, the occurrence of delayed bleeding casts doubt on the efficacy of ligature of feeder vessels in circumstances where the malformation can be excised.

TABLE VI.—Distribution of 110 Bleeding Aneurysms (Angiography)

Anterior cerebral artery:		
(a) first part	1	
(b) at anterior communicating	33	
(c) distal part	1	
Carotid artery:		
(a) at posterior communicating	49	
(b) bifurcation	2	
Middle cerebral artery	18	
Basilar	5	(2 fusiform)
Posterior cerebral	1	
Patients with multiple aneurysms	10	

TABLE VII.—Grading of Cases—(Botterell)[1]

1. Conscious.
2. Drowsy without significant neurological deficit
3. Drowsy with significant neurological deficit an possibly with clot.
4. Major neurological deficit deteriorating with clo or old patients with less severe deficit bu degenerative vascular disease.
5. Moribund or near moribund with failing vita centres and with extensor rigidity.

Ruptured Intracranial Aneurysm

The distribution of 110 bleeding aneurysms is summarized in Table VI.

The results of surgical treatment of ruptured aneurysms depend in large part on the site of the aneurysm, the clinical state of the patient and the interval between the hæmorrhage and operation. Thus local factors such as cerebral swelling with retraction for deep exposure and arterial spasm adjacent to a clip are more important in the relatively small vessels of the anterior communicating region than in carotid aneurysms. Another major factor bearing on the outcome is the preoperative state of the patient, in terms of level of consciousness, associated intracerebral clot or ischæmic softening, age, and the presence of degenerative cerebro-vascular disease. Botterell *et al.*[1] have summed up these variables as they bear on the individual patient by a system of grading (Table VII).

It is well known that the risks of an intracranial procedure are greatly lessened by an interval of two weeks or more from the time of the last hæmorrhage. Unfortunately, this interval covers the period marked by high incidence of recurrent bleeding and many patients would be lost or crippled while awaiting a safer time for operation.

In a study of the prognosis in subarachnoid hæmorrhage Höök[2] found mortality to be the highest on the first day after hæmorrhage and 33% of the patients died within two weeks. After two weeks the mortality decreased appreciably and

TABLE VIII.—Surgical Methods

Indirect (internal carotid ligation)		2
Direct—		
clip or ligature	60	
muscle pack	4	
gauze filigree	2	
proximal carotid clip	2	
	68	
Total		70

was 20% after eight weeks. In Walton's series,[3] 91% of the deaths resulting from the first hæmorrhage, and 61% of the deaths from recurrent bleeding occurred within two weeks of the initial hæmorrhage. The peak incidence of recurrent bleeding was in the second week. Because of this fact, it has been our policy to treat these cases as urgent problems with immediate angiography and early operation.

Seventy patients shown to have an aneurysm by angiography were subjected to an operative procedure. Forty-eight (70%) were operated upon within one week of the last hæmorrhage and 36 (51%) were explored within 48 hours.

In all but two instances a direct attack on the lesion was undertaken (Table VIII).

An effort was directed towards obliteration of the aneurysm by silver clip or by a ligature. The neck of the aneurysm was considered unsuitable for clipping in eight patients who are discussed under their respective categories. All but four of the surviving patients with a clip or ligature on the aneurysm were subjected to postoperative angiography. The aneurysm was shown to be obliterated except in two patients with middle cerebral artery aneurysms which produced fatal recurrent hæmorrhage in 10 days and 24 hours respectively. An Olivecrona clip slipped off the neck in the first case and two ligatures had rolled down the fundus of the aneurysm in the other.

In this regard it is important to place a clip fully across the neck or fundus of an aneurysm for, as has been seen in the operating room, those placed part way across have a tendency to slide off slowly with the pulsations of the proximal portion of the lesion. Great care must be taken when using a ligature on the neck of an aneurysm arising from a small parent vessel. Tightening the knot at the very base of the sac may produce a kink in the artery with resultant stenosis or even occlusion of this vessel. Three postoperative deaths in this series were a direct result of massive cerebral softening from kink-producing ligatures on the necks of large anterior communicating and middle cerebral aneurysms.

The surgical attack on aneurysms has been conducted under hypothermia since 1955. Of 24 patients operated upon without hypothermia, 12 died. This excessive mortality of 50% was largely the result of operations on sick patients with ruptured anterior communicating aneurysms. In contrast, 46 patients cooled to 27° to 30° C., were operated upon with 11 deaths, a 24% mortality (Table IX). This improvement resulted in large part from reduction of cerebral swelling and control of hæmorrhage from aneurysmal rupture at operation. In addition a major feature has been the routine use of brief periods of occlusion of the carotid and occasionally the vertebral arteries in the neck lasting four to eight minutes. This has allowed swift completion of the dissection of the slack aneurysmal sac and placement of a clip without the same danger of rupture with frantic attempts to control bleeding.

TABLE IX.—Artificial Hypothermia

	Patients	*Deaths*	*Percentage*
Not used.....	24	12	50
Used.....	46	11	24

Carotid Aneurysms

Two patients had ruptured carotid bifurcation aneurysms. One was clipped successfully—the other tore at its base when clipped and the bleeding was controlled finally by a clip on the carotid just below it. A permanent hemiplegia occurred on the second postoperative day.

Of 30 ruptured carotid-posterior communicating aneurysms, 23 were approached directly with eight deaths. Four deaths followed attempts to save three Grade 5 and one Grade 4 patients. Four Grade 3 patients died as a result of errors in judgment and technique. Two deaths occurred from recurrent bleeding as a result of slipping of narrow Olivecrona clips. In another patient, a second "security" clip nudged the first and tore the neck of the sac. The bleeding was controlled with muscle but fatal recurrent hæmorrhage occurred the following day. The fourth patient with two carotid aneurysms died suddenly a few hours after operation. The autopsy disclosed that a Mayfield clip had finally torn the base of the previously unruptured short sessile aneurysm which lay under the optic

TABLE X.—Carotid Aneurysms

Risk	0—48 hours		2—7 days		7—14 days		over 14 days	
	No. of patients	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*
1.....	2	0	4	0	2	0 (1p)	6	0
2.....	1	0	1	0			1	0
3.....	7cc	3	1	0	2c	2	1	0
4.....	1c	1c			0	0		
5.....	3c	3c						
Totals.....	14	7	6	0	4	2	8	0

Legend: c—intracerebral clot in one case. Total of 32 patients: 9 deaths
p—poor result 1 poor result

nerve at the origin of the ophthalmic artery. Three aneurysms considered unsuitable for clipping were packed with hammered muscle. One Grade 5 patient died a few hours later. The other two have remained well for one and three years. In another case it was possible to insert a piece of muscle into the aneurysmal sac through a large rent in its neck caused by manipulation. Postoperative angiography failed to fill the aneurysm and the patient has remained well for five years.

The two carotid ligations were performed in this group. One 63 year old hypertensive woman with a non-protein nitrogen of 63 mg. % died from recurrent bleeding the day after a Selverstone clamp was completely closed. The aneurysm in the other patient was shown by angiography after internal carotid ligature in the neck, to be thrombosed.

As a group, carotid aneurysms are the most satisfactory to attack directly. Excellent exposure of the neck of the aneurysm is obtained by a subfrontal approach along the sphenoid ridge using a minimum of retraction. The ipsilateral common carotid artery may be compressed for brief periods by the anæsthetist under the drapes during the dissection of the neck of the aneurysm and the application of a clip. Unlike the anterior cerebral-anterior communicating region, parent arterial spasm did not prove to be the same postoperative hazard. It occurred in only one case on the fifth postoperative day with a sudden hemiplegia, proved by angiography to accompany massive spasm of the carotid siphon.

Anterior Cerebral—Anterior Communicating Aneurysms

Table XI summarizes the results of treatment in 24 cases.

All but two were found in the anterior communicating region. One large proximal anterior cerebral aneurysm was trapped between clips and one distal aneurysm was clipped after removal of an interhemispheral subdural clot, both without incident. Nineteen anterior communicating aneurysms were clipped at the neck with seven deaths. One short sessile aneurysm in a hypertensive patient was considered unsuitable for clipping and was packed with hammered muscle. Death followed recurrent hæmorrhage three weeks later. Two patients succumbed with massive bi-

TABLE XI.—Anterior Cerebral—Anterior Communicating Aneurysms

Preoperative risk	0—48 hours		2—7 days		7—14 days		over 14 days	
	No. of patients	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*
1.....	1	0					4	1
2.....	3c	0	2	1 (1p)				
3.....	3	0 (3p)	1	1	1c	0	1	0 (1p
4.....	3	2						
5.....	3cc	3	2	2				
Totals.....	13	5	5	4	1	0	5	1

Legend: c—intracerebral clot Total of 24 patients: 10 deaths
p—poor result 5 poor results

frontal softenings consequent to kink-producing ligatures on the necks of large anterior communicating aneurysms.

Early operation on Grades 4 and 5 patients was uniformly unsuccessful in preserving useful life and merely caused further damage to an already critically injured brain. The only survivor among eight cases was classified as "poor" because of continuing dementia. On the other hand, of ten early operations in the better risk groups, only two patients died but results in three Grade 3 patients were classified "poor". Both deaths were the result of delayed cerebral swelling (two and three days) which was not controlled by removal of the bone flap and subtemporal decompression. Urea was not in use at the time but it may well prevent or control this problem in many instances.

The anterior communicating region is the most difficult to expose in a satisfactory yet safe fashion. Firm, deep retraction and removal of the postero-medial corner of the orbital surface of the frontal lobe is often required. Adjacent are the depths of the frontal lobes and anterior hypothalamic region, irrigated by the central branches of the anterior cerebral arteries. Stretch on these tiny vessels and parent arterial spasm particularly during early operation on very sick patients, may result in bilateral frontal and deep cerebral softening and swelling which may be incompatible with life or render the patient stuporous or demented. To facilitate good exposure with gentle retraction, an effort must be made to obtain a slack brain through some combination of intravenous urea, hypothermia and lumbar or ventricular drainage of cerebrospinal fluid. The concomitant use of papaverine, procaine[4] and phentolamine[5] on the exposed vessels apparently minimizes arterial spasm.

Middle Cerebral Aneurysms

The results of treatment in 11 cases of ruptured middle cerebral aneurysms are indicated in Table XII. Of importance is the fact that nine of the 11 aneurysms had produced a significant intracerebral clot, temporal or temporo-parietal, with a deep frontal extension in one case. The presence of a clot can usually be forecast by the elevation of the middle cerebral leash of vessels. This should not be a deterrent to operation. Seven of nine patients with intracerebral bleeding survived and only one was considered a poor result from a pre-existing neurosis and intractable headache. The aneurysms arose at the trifurcation of the artery and lay on the insula in 10 cases. They were exposed by removal of the anterior portion of the temporal operculum. Evacuation of clot not only provided room but often left most of the dissection of the aneurysmal sac complete.

One patient, under treatment for subacute bacterial endocarditis, developed a massive temporo-parietal clot from a rupture in one of the deep ascending parietal branches. The bleeding artery was clipped but unfortunately the segment was not excised. It was presumed to be a ruptured mycotic aneurysm. Two patients with temporal clots had large aneurysms with broad necks involving the trifurcation. These were treated by wrapping the whole of the aneurysmal sac with surgical gauze, gathered and tied at the neck. There has been no recurrent bleeding from the reinforced sacs and the patients have remained well for five and eight years respectively.

TABLE XII.—Middle Cerebral Aneurysms

	0—48 hours		*2—7 days*		*7—14 days*		*over 14 days*	
Risk	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*
1							1	0
2	1	1					1c	0
3							2cc	1
4	4cccc	0p	1c	0				
5	1c	1						
Totals	6	2	1	0			4	1

Legend: c—intracerebral clot
p—poor result

Total of 11 patients: 3 deaths
1 poor result

TABLE XIII.—Basilar Aneurysms

Site	*Risk*	*Result*
At superior cerebellar artery	3	well
Bifurcation	3	R. field defect
Bifurcation	4	died

The remaining eight aneurysms were occluded by a long clip or ligature. There were three postoperative deaths, one of which was clearly avoidable. In this patient recurrent hæmorrhage occurred 24 hours after a large sac had been deliberately opened and then occluded by two silk ligatures at the base. Re-exploration revealed that the ligatures, still intact, had been rolled down the limp sac by the proximal pulsations to a point just beyond the original rent. The patient died seven days later from massive softening of the hemisphere and it was discovered that the new ligature had been tied too firmly, kinking and stenosing the middle cerebral artery. It would seem wise to keep the basal ligature a little slack and use a transfixing suture just beyond on large sacs which have been deliberately opened. Another patient remained well until the seventh day before developing fatal massive swelling of the hemisphere. The third death followed an operation on a Grade 5 patient.

Basilar Aneurysms

Three patients with repeated bleeding from ruptured basilar aneurysms have been operated upon within 24 hours of the last hæmorrhage (Table XIII).

Two aneurysms arose from the top of the basilar bifurcation and one from the anterior aspect of the basilar artery at the origin of the superior cerebellar arteries. It was possible to occlude the neck in each case by an Olivecrona or Mayfield clip. As few of these aneurysms have been attacked directly, they are to be discussed as a separate problem in another paper.

Table XIV summarizes the results of surgical treatment in 70 cases with reference to the grading of the patient and the time interval after the last hæmorrhage. It is clear that the highest mortality and morbidity result from early operation on Grade 4 and Grade 5 patients. Six Grade 5 patients were operated upon because of known associated intracerebral clot. All died, although two were subjected to preliminary hypothermia for two and four days. Grade 4 patients gravely ill with cerebral softening and swelling are also poor risks. However, when harboring a demonstrable intracerebral clot, it is worthwhile to evacuate the clot and obliterate the aneurysm, for three of five such patients survived.

It should be recognized that surgical treatment, aside from evacuation of clot, is solely for the prevention of recurrent bleeding and may aggravate a critically swollen brain by deep retraction and promotion of further vascular spasm. Early operation, therefore, should not be considered for stuporous patients not harboring a clot. This is particularly true with ruptured anterior communicating aneurysms, for Birse and Tom[6] have shown the frequent occurrence of multiple and widespread cerebral infarctions in the brains of fatal cases, whether they were operated upon or not. Survival may be aided by careful nursing and possibly prolonged hypothermia to surmount the stage of acute cerebral swelling and softening. In many of the survivors, a persisting profound neurologic defect will militate against a delayed operative procedure.

TABLE XIV.—Bleeding Aneurysms—Results of Operation—70 Cases

	0—48 hours		*2—7 days*		*7—14 days*		*over 14 days*	
Risk	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*	*No. of patients*	*No. of deaths*
1	3	0	4	0	2	0	11	1
2	5	1	3	1			2	0
3	13	4	2	1	2	1	4	1
4	6	3	1	0	1	1		
5	6	6	2	2				
Totals	33	14	12	4	5	2	17	2

Basilar aneurysms—3 cases under 24 hours—1 death

Total of 70 patients: 23 deaths: 9 poor results

On the other hand in the presence of less severe bleeding and brain injury, early operation (<1 week) in 30 Grade 1 to Grade 3 patients resulted in seven deaths (23%) and results in five cases were classified as poor. Three of the seven deaths were due to avoidable technical errors and three of the five poor results were in Grade 3 patients with anterior communicating aneurysms. Unfortunately no comparison is possible with a similar group treated conservatively but there is no reason to believe that the results would be improved. Of importance is the fact that patients with surgically obliterated aneurysms can lead normal lives free from the dread of recurrent bleeding. Our increasing knowledge of the causes of the operative and postoperative catastrophes and their prevention will surely lower the mortality and morbidity figures considerably. It seems therefore, that patients not critically ill and those with a clot, should continue to be subjected to early angiography and operation. The security from recurrent bleeding and restoration of cerebral function makes the risk of early operation much less than that to be expected from conservative management.

Summary

The results of investigation of 189 patients with spontaneous intracranial bleeding are reviewed. Fatal recurrent bleeding occurred in 22% of patients with negative angiography. Sixteen of 21 arteriovenous malformations were operated upon with no mortality or increased morbidity. Seventy of 110 ruptured aneurysms were treated by direct surgical management, 70% within one week of the last hæmorrhage and 50% within 48 hours. The conclusion was reached that patients with ruptured aneurysms, not critically ill and those known to have a clot should continue to be subjected to early angiography and operation.

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Résumé

Les chances de survie après une hémorragie intra-crânienne (sous-arachnoïdienne) spontanée sont tellement compromises que l'intervention d'urgence doit être considérée dans un grand nombre de ces cas. Combien regrettable n'est-il pas d'observer des malades survivre à une première hémorragie pour succomber de façon inattendue quelques jours ou quelques semaines plus tard à une nouvelle hémorragie. De plus, l'intervention semble s'imposer pour conserver la vie et l'activité cérébrale dans le grand nombre de cas où un caillot intra-cérébral demeure.

La prévention de l'hémorragie récurrente est devenue un problème chirurgical de majeure importance, particulièrement dans les cas d'anévrismes rupturés, qui sont les plus souvent en cause, et les plus difficiles à contrôler. Même si l'oblitération de l'anévrisme est habituellement possible, son exposition qui requiert une rétraction profonde peut entraîner une série de phénomènes désastreux qui conduisent à l'œdème et au ramollissement cérébral massif, entraînant alors une altération profonde de la ponction cérébrale ou la mort. Malgré cela, l'intervention assure un taux de mortalité inférieur à celui de 50% qui est celui du traitement conservateur. C'est avec l'idée que la plupart des anévrismes sont petits et accessibles, qu'on se doit de rechercher d'améliorer la technique chirurgicale afin de pouvoir éliminer d'emblée la possibilité d'hémorragie récurrente.

Cette étude comprend 189 malades qui ont cliniquement été considérés, avant angiographie, comme ayant saigné d'un anévrisme ou d'un angiome intra-crânien. Seize cas d'angiome et 68 d'anévrisme furent traités d'emblée chirurgicalement: 70% de ces derniers ayant été opérés moins d'une semaine après la dernière hémorragie et 50% dans les 48 heures.

L'hémorragie sous-arachnoïdienne fut vérifiée dans tous les cas par ponction lombaire, et la distribution vasculaire démontrée précocement par une angiographie carotidienne bilatérale, complétée chez 30 malades dont l'angiographie carotidienne était négative, d'une angiographie vertébrale. (Tableaux I et II)

En deux occasions, un anévrisme fut démontré en répétant l'angiographie, d'où l'importance d'angiogrammes de contrôle après deux ou trois semaines, ou du moins, avant le congé du malade.

Une hémorragie récurrente fatale est survenue chez 22% des malades dont l'angiographie était négative. Seize des 21 cas de malformation artério-veineuse furent opérés sans mortalité ni morbidité accrue.

Il ressort, en conclusion, que les malades présentant un anévrisme rupturé et qui ne sont pas moribonds et ceux que l'on sait porteurs d'un caillot devraient continuer d'être soumis à l'angiographie précoce et à l'opération.

THE BENIGN "PHYSIOLOGICAL" BREAST DISEASES: A PLEA FOR CONSERVATIVE MANAGEMENT*

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MUCH DISCUSSION, both in the written and spoken word, has kept physicians and surgeons well aware of the controversies currently encountered in the treatment of breast cancer. In contradistinction, it seems odd that so little emphasis has been placed upon reappraisal of the therapeutic situation in benign breast disease, a process affecting a far greater number of patients, but in which few clear-cut plans for management are commonly in use. It is for this reason that repetitive emphasis upon conservation in the care of the benign process seems worthwhile.

This problem was reviewed previously[1] with emphasis on the fact that definite proof of a pre-cancerous tendency in benign breast disease was clearly lacking. Consequently, surgical mutilation appeared unwarranted.

STATISTICAL REVIEW

In this appraisal[1] of prospective studies of benign disease (including both chronic mastitis and its papillomatous intraductal variants) the incidence of subsequent cancer after local excision of a benign process appeared no greater than that to be anticipated in the general population. Retrospective studies of malignant disease similarly did not demonstrate an increased incidence of previous treatment for benign disease in the patient's past history. It was pointed out in addition that, although recurrence of symptoms after treatment of benign disease might occur in approximately 15% of cases, unilateral simple mastectomy was not an adequate solution even in this problem since, as might be anticipated, almost 50% of these recurrences affected the opposite breast.

Although recurrence of symptoms might be annoying it was, of course, the fear of subsequent cancer which led to the attempt to prevent this catastrophe by utilizing simple mastectomy as a prophylactic gesture, particularly in those diseases in which epithelial hyperplasia represented the outstanding feature of the microscopic picture. Once again the futility of such an approach was stressed with the observation that subsequent cancer was just as likely to occur in the opposite breast as in the breast under treatment. In the absence of any definite proof of a marked increased incidence of subsequent cancer, the physical and psychological mutilation of a bilateral mastectomy, which remained the only logical extension of aggressive surgical therapy, could scarcely be considered.

Subsequent literature has continued to provide support for this conservative outlook. In the statistical analyses previously reported[1] the occurrence of subsequent cancer after local excision of benign disease varied between 1% and 2% as compared to a loosely calculated incidence in the general population of 0.5% to 1%. Recent prospective studies have also failed to show any evidence of an increased incidence of subsequent cancer. Rosemond *et al.*[2] report 150 cases with cystic disease treated by aspiration in which only two subsequently developed cancer, one of which occurred in the opposite breast. Two of their patients had cancer in the cyst wall but the presence of a residual mass led to excisional therapy with immediate recognition of the associated malignant process. Care is obviously necessary in ascertaining that the lump actually disappears after aspiration. Hendrick[3] also notes 484 patients having local surgical treatment for benign disease in which group only four subsequently developed cancer, an incidence well within the range noted above. In his series, 167 had cysts aspirated with no case of late cancer recorded, and in this group seven with residual induration after aspiration showed no evidence of cancer when the area was excised.

Retrospective studies similarly provide the same kind of basic factual information. Cairncross[4] records previous cystic disease present in only 0.6% of 1544 cases of breast

*Presented at the Annual Academy Day, London Academy of Medicine, London, Ontario, April, 1959.

cancer treated at the Presbyterian Hospital, New York City. Hendrick[3] reports in addition 452 cases of cancer of the breast, in which group only four gave a previous history of removal of areas of benign disease. These incidences are well within the percentage range for a comparable group in the general population.

In the case of the papillary variants of benign breast disease the identical pattern continues in evidence. Hendrick[5] notes 208 cases of intraduct papilloma or benign papillary cystic disease, in which group after local treatment of the benign process only two cancers subsequently arose, both in the opposite breast. Madalin, Clagett, and McDonald[6] also record approval of the conservative treatment of nipple discharge particularly in the absence of a palpable mass.

It would appear, therefore, that as these large series of cases are carefully evaluated, and as surgeons gather thereby a wider knowledge of these benign processes, the total experience, both in the practical management of the patient and in the academic appraisal of results achieved, leads inevitably to the establishment of a plan of treatment for benign disease in which conservatism must remain the fundamental keystone.

In order to apply conservative principles properly and create practically effective therapy, one must clearly understand the basic physiology of the breast and the pathophysiology which leads to the exhibition of benign disease processes. These normal and abnormal physiological processes will now be discussed briefly.

Normal Physiology of the Breast (and Uterus)

The completely normal physiological changes produced in the breast during a normal menstrual cycle are diagrammatically represented in Figs. 1 and 2. Two features are of importance in this regard.

In the first place, as has now become accepted teaching, it would be illogical to expect a perfectly symmetrical stimulation and regression of breast tissue during each of these successive monthly cycles for the entire duration of the patient's sexual life. Irregularities must be anticipated and if they occur in localized areas they will produce the breast lumps which create one of the presenting problems of benign disease.

In the second place, the breast changes only occur in the presence of a normally functioning pituitary, and the inter-relationship between the ovary and the pituitary must be perfectly adjusted for normal breast and uterine changes to develop in the proper sequence. Experimentally, using hypophysectomized, adrenalectomized, and castrated animals, œstrogen and progesterone alone have no effect on the breast, although œstrogens will produce mitotic epithelial changes locally. Purified prolactin will carry the change only to mid-menstrual stages and both adrenocorticotrophic hormone and growth hormone are necessary in addition if full mammary development is to occur.[7] In such a complicated relationship aberrations are logically to be anticipated, and as might also be expected, since the breast and uterus respond in similar fashion as target organs for the same hormonal substances, one would logically assume that these aberrations would frequently be exhibited by correlated changes occurring simultaneously in these organs.

Abnormal Physiology of the Breast (and Uterus)

The thesis that the breast and uterus should have interrelated diseases is supported by the frequent association of mammary and uterine complaints. It has long been recognized, of course, that benign breast disease tends to occur most often in patients with menstrual irregularities and a history of sterility. There is now confirmatory evidence that the entire pituitary-gonad-thyroid axis is unbalanced in these conditions.

On the purely histological plane Martel and Sommers[8] who separate benign disease into adenofibrosis and chronic cystic mastitis (as we have also done in the past,[1] utilizing the more descriptive terms of non hyperplastic and hyperplastic disease respectively) show the frequency with which changes in these endocrine relationships may be demonstrated. They have dis-

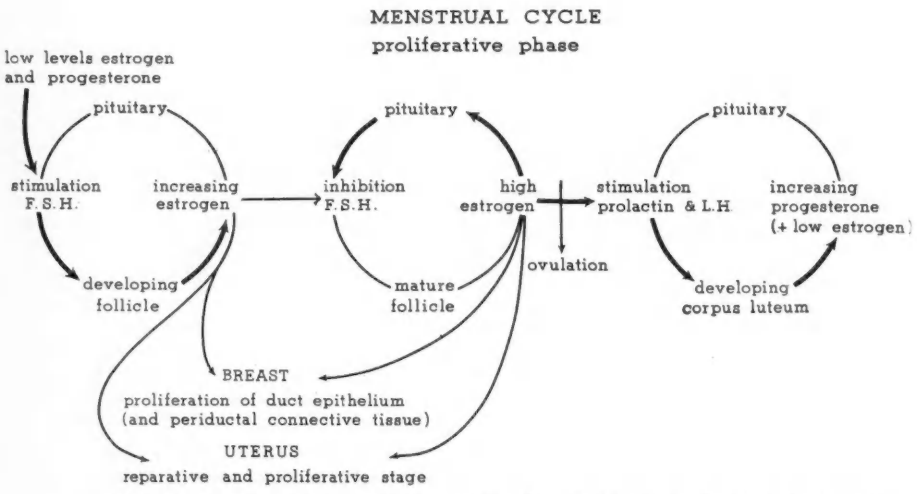


Fig. 1.—Diagrammatic representation of physiological changes produced in the breast during the proliferative phase of the normal menstrual cycle.

covered ovarian cortical stromal hyperplasia to be present in 19 of 20 cases of chronic cystic mastitis and in 17 of 20 cases of adenofibrosis, with the change less apparent in this latter group who are considered potentially to be in an early stage of the fully developed process of chronic cystic mastitis. They point out that the fact that the ovarian stroma produces œstrogen is well authenticated. Associated with the ovarian stromal overgrowth there is a pituitary basophilic hyperplasia. These pituitary cells release the gonadotrophins which produce, in addition to ovarian stromal hyperplasia, the proliferative hyperplastic uterine endometrial changes recorded in 10 of 14 cases of chronic cystic mastitis and 10 of 18 cases of adenofibrosis. This again indicates the close relationship between diseases of the breast and uterus, and also the

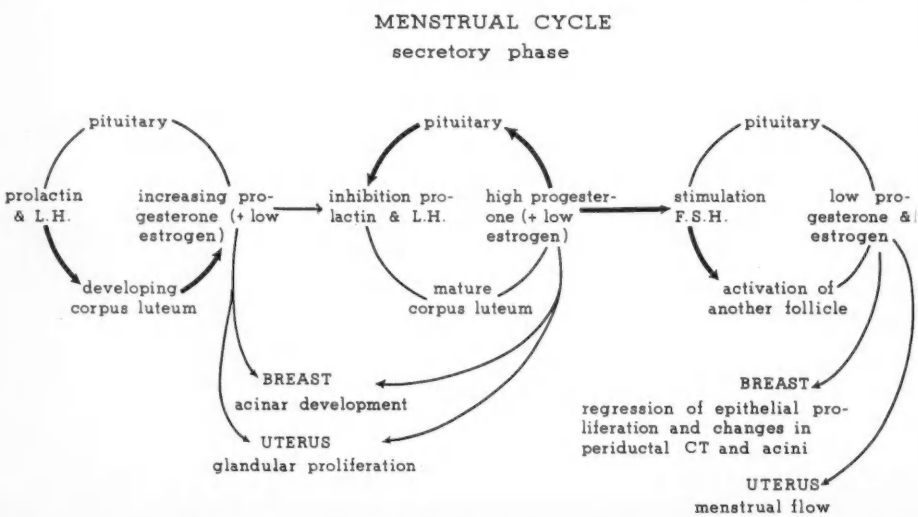


Fig. 2.—Diagrammatic representation of physiological changes produced in the breast during the secretory phase of the normal menstrual cycle.

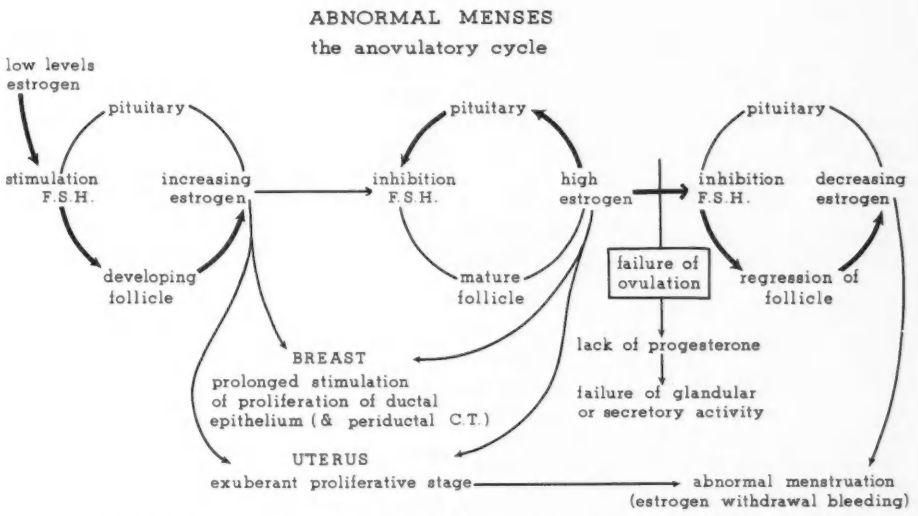


Fig. 3.—Diagrammatic representation of changes produced in the breast during an anovulatory menstrual cycle.

earlier changes to be detected in adenofibrosis. As an additional observation the thyroid was found to be abnormal in 11 of 20 cases and in these cases there was a subnormal number of pituitary amphophil cells, which cells are now known to be the source of the thyrotrophic hormone.

From the clinical and laboratory aspect of the problem additional evidence is also present to indicate the importance of this general endocrine abnormality. It has become apparent that, in the presence of any abnormality affecting ovarian function either in the nature or timing of the trophic stimulation or in the ovary itself, failure of ovulation is the first change to be detected, since in the transformation of the follicle to a corpus luteum, a delicately balanced hormone relationship is essential. The failure to produce functioning luteal bodies results in menstrual changes characteristic of the anovulatory cycle. This effect may be due to an intrinsic defect in the ovary or more probably due to a more fundamental defect in the process of œstrogen stimulation of the pituitary release of luteinizing hormone and prolactin. The nature and results of this anovulatory cycle are depicted in Fig. 3. In this case the failure to produce luteinizing hormone results in a deficiency of progesterone and, therefore, prevention of the normal maturation in the breast and uterus from the proliferative to the secretory phase of the monthly cycle. Consequently stimulation is continued, as indicated by ovarian stromal activity, until progressive inhibition of the follicle stimulating hormone removes the trophic stimulus for continuing survival of the follicle. At this stage, œstrogen levels fall, and the proliferative changes both in the breast and the uterus, which are now no longer supported by this hormonal stimulus regress with associated uterine bleeding of œstrogen-withdrawal type. Since this bleeding may be a comparatively regular cyclic process, and similar in amount and duration to normal menstruation, the patient may well be unaware that the menstrual cycle is abnormal in any way.

However, apart from the sterility which is of necessity associated with the failure of ovulation there are other indications of the frequency with which this situation is encountered. Premenstrual endometrial biopsies will show proliferative change with none of the glandular secretory activity which is dependent upon progesterone stimulation. In keeping with the lack of progesterone, which is normally metabolized in the liver, with approximately 20% to 30% excreted in the urine as pregnanediol, there will be an absence of this substance (pregnanediol) in the urine and

also a low progesterone content of the breast tissues themselves. These patients show, in addition, a monophasic temperature record in comparison with the normal diphasic temperature response during a normal menstrual cycle.

"Physiological" Breast Diseases

Although progesterone deficiency is then the fundamental physiological link in these processes, the œstrogenic stimulus, continuing in relatively high concentration, represents for practical purposes the actual "cause" of the diseases under consideration. The fact that benign breast disease does not occur in patients castrated before puberty and tends to regress when ovarian function is controlled at the time of a spontaneous or induced menopause, indicates the significance of this statement. Womack[9] points out that chronic cystic mastitis has been produced in both man and animals by excessive œstrogen stimulation and never without œstrogen. It becomes evident, therefore, that in the conservative management of benign breast disease, attention must logically be directed to minimizing the effect of this relative excess of endogenous œstrogen.

Endogenous œstrogen may produce a physiological mammary response in two ways, and in so doing, may create pathophysiological breast changes which naturally fall into two main clinical groups, although both are clearly related since dependent upon the identical basic stimulus. We have chosen to term these processes the non hyperplastic and hyperplastic diseases while recognizing their relationship as well as the suggestive evidence recorded above, that the first type may in some instances represent a precursor or early stage of the second type of disease.

Non Hyperplastic Disease (Adenofibrosis)

These changes result from the indirect effect of œstrogen on the breast as a result of its systemic biochemical action as a steroid in producing increased renal tubular reabsorption of sodium (and water) with general increase in extracellular fluid volume. This increase is possibly most noticeable in the normal target organs of œstrogen stimulation. At any rate, the increased extracellular fluid volume tends to persist and increase throughout the latter part of an anovulatory cycle, since there is no normal compensatory diuresis as a result of the progesterone release, which occurs in the last half of a normal menstrual cycle. This increasing "retention" produces painful swelling in the breast, which is normally a firm organ unsuited to volume alteration and well supplied with delicate sensory nerves which are stimulated by the attendant pressure effect. In addition, as is customary in interstitial tissue response throughout the body, the increased extracellular fluid volume causes the reactionary productive fibrosis in the extrinsic breast stroma, which may eventually develop so remarkably that the entire breast becomes quite hard and clearly delineated from the surrounding subcutaneous adipose tissue. The association of premenstrual pain with a hard saucer-like breast has generally been termed adenofibrosis. As might be anticipated, since the symptoms are due to an œstrogen-induced fluid and sodium "retention", they tend to subside rapidly and dramatically with the sudden fall in œstrogen levels that promotes the onset of œstrogen-withdrawal bleeding.

Hyperplastic Disease (Chronic Cystic Mastitis)

In this case the breast changes result from the direct local effect of œstrogen stimulation of the breast itself. With the relative increase in the concentration of œstrogen and the prolongation of its period of effective stimulation, there will be a marked increase in the degree of proliferation of intraductal epithelium and the intrinsic periductal connective tissue of the breast. Consequently, the breast tends to become ropey and nodular and, as a result of the increased number of cells sloughed into the lumen of the ducts to be liquefied by autolytic digestion, there is an increased accumulation of fluid and debris within the duct which may be forced to the nipple by pressure as a non puerperal discharge. The discharge tends to appear from several duct orifices, is often bilateral, and although occasionally a clear straw colour

in appearance, it usually has a murky or pasty nature.

The previous report[1] emphasized the fact that gross cysts (which are due to hygroscopic increase in the amount of autolyzed intraductal cellular debris) and duct papillomata (in which the epithelial hyperplasia demonstrates vascular cores) may conveniently be considered as variants of this hyperplastic mammary response.

Premenstrual Tension

Both the processes described above are frequently associated with the symptoms of premenstrual tension. Current opinion makes this a logical association since it is now felt by most observers that this syndrome, often so disabling during the premenstrual week or ten days, is largely due to the same œstrogenic stimulation of increased sodium and water reabsorption that results in general systemic increase in the extracellular fluid volume. The abnormal premenstrual weight gain in association with significant rises in diastolic and systolic blood pressure in those women with severe complaints support this contention that an extracellular fluid volume increase is the basic phenomenon here. Mazer and Israel[10] demonstrated the high level of œstrogen present during this period many years ago. Much information is now available concerning the effectiveness of measures designed to alleviate the troublesome symptoms of this syndrome, and because it is presumably produced by the very factors which initiate benign breast disease, this information should be directly applicable to the management of the problem under discussion here. Bickers[11] has reported marked improvement in 22 of 49 patients with premenstrual tension who complained primarily of breast fullness, headaches and abdominal bloating in which group, treatment with a diuretic (chlorothiazide) was started at the onset of premenstrual symptoms and continued through the first day of menstruation. The average weight gain during the treated cycle showed a remarkable fall (from 5.25 lb. to 1.75 lb.) and 20 of the 22 patients were remarkably improved. In the entire series of 49 patients only 11 failed to obtain benefit and it was felt that this was due to the fact that they were refractory to the diuretic used, since the weight loss was not as dramatic as that noted above.

Other measures utilized in the management of this syndrome, although usually based on an apparently valid rationale, have failed to produce such gratifying responses. Attempts, for example, to hasten œstrogen metabolism in the liver by administering vitamin B complex in order to maintain hepatic function at a high level of efficiency, have provided little subjective improvement, although perhaps capable of supporting the patient's general sense of well being.

More recently attempts have been made to correct the basic progesterone deficiency by using synthetic progestational steroids with a view to promoting a more normal menstrual cycle (even in the absence of ovulation). These agents, if effective, should overcome the increase in extracellular fluid volume by their action in increasing urinary excretion of sodium[12] and, of course, when supplementing the local effect of œstrogen on the breast and uterus, promote the development of a normal secretory phase. Whether or not they may, by their ability to depress pituitary luteinizing hormone, when a series of therapeutic courses is discontinued result in release of this luteinizing hormone by a rebound phenomenon with stimulation of ovulation, corpus luteum formation and spontaneous return to an ovulatory cycle, is highly theoretical. Since other factors than symptomatic relief alone have to be considered here, the period over which these agents should be given requires much thought. There seems little doubt but that they are capable of preventing ovulation in normal cycles due to this depression of luteinizing hormone,[13-15] and actually these agents are now being studied as potentially effective contraceptive measures. If one is dealing with a primary anovulatory cycle and sterility is already an established fact, one may reasonably resort to such therapy in severely disabling instances of premenstrual tension, but prevention of ovulation can scarcely be a proper emphasis in other cases. The fact, however, that ovulation occurs on discontinuing the drug may permit its use on

a temporary basis in order to establish a more normal physiological sequence. It is generally suggested that therapy be started at approximately the 15th day in order not to inhibit ovulation, if it is to occur, and continued through to the 25th day in order to produce progesterone-withdrawal bleeding at about the time when normal menstrual flow might be expected to occur. In some cases, it may be started in the pre-ovulatory period in order to effect a gradual increase in circulating levels, much as occurs in the last half of the normal cycle.

Womack[9] has recently discussed the effectiveness of a long acting agent of this type (hydroxy-progesterone caproate, 250 mg.) given only once a month by intramuscular injection to patients with mammary complaints, and he reports that in 41 patients premenstrual breast pain, nipple discharge and cyst filling usually improved within three months. This indicates again that the same principles quite properly underlie the management of these related disorders.

Management of Benign "Physiological" Breast Disease

As outlined in the previous report[1] benign disease, of the type predicated on abnormal hormonal stimulation, is exhibited clinically in three common ways; a lump in the breast, a nipple discharge, or discomfort in the breast of varying degree during the premenstrual period. Mastodynia, when used in reference to mammary pain that is not definitely premenstrual in occurrence, is coloured by psychogenic attributes and is not properly under discussion in this analysis.

The treatment of the discrete lump (including gross cystic disease) was thoroughly reviewed previously[1] and mention will be made only of the continuing emphasis on conservatism within the limits of which emphasis, one should avoid any major mutilation by advising local resection of these lumps and aspirations only in the case of cysts, provided they disappear completely after being emptied. Residual palpable induration after aspiration must, of course, be excised for pathological appraisal.

Nipple discharge is handled in similar fashion. If the discharge is bloody and apparently from a single duct, there is a 15% to 20% chance that it is due to an intraduct carcinoma, even in the absence of a palpable mass, and consequently local excision by retrograde cannulation of the duct from its nipple orifice is indicated. If the discharge is produced from several ducts and particularly if it is *not* sanguineous one may be certain that a benign hyperplastic process is involved in its development, and a conservative plan is, therefore, indicated according to the principles to be outlined below. Observation is, of necessity, advisable in order to assess the results of the therapy and to make certain that an unrelated lump does not subsequently appear in these relatively overstimulated breasts.

It is in the management of premenstrual pain, however, that the physiological principles enunciated above have clinical application within the basic principles of a conservative approach to the problem. Certainly this symptom is one of the most difficult for the physician to treat adequately in view of the continuing abnormal physiological stimulus responsible for its presence, and the unfortunate and hazardous emotional reactions on the part of the patient to whom such a complaint has only one real significance, namely that of indicating "cancer". It is necessary, therefore, to create symptomatic relief, in order to have some concrete basis for providing satisfactory reassurance for the patient. The following plan of treatment is one designed to afford such relief within the framework of the conservative approach recommended in these instances.

Treatment Programme

As in all therapeutic plans treatment begins most commonly with the measures having the least effect on the patient as a whole and progresses subsequently to measures which affect the host in a more serious way only if necessary in view of the inadequate response to the initial therapy. It is in this light that the following sequence should be viewed.

"*Support*" is the key-note of the programme; psychological support to provide emotional stability for the patient, physical support for the heavy, swollen, tender breasts, and physiological support designed

to control the systemic and local reaction to the relative œstrogenic excess. In this last instance diuretics are used to minimize the increase in extracellular fluid volume and in uncontrolled cases an attempt can be made to increase the metabolism of œstrogen using vitamin B_1 or to counterbalance its effect by exhibiting progesterone.

1. *Psychological Support*

Not infrequently this is the only measure indicated in intelligent women for whom a careful, meticulous explanation with kindly reassurance has meaningful quality. A complete physical examination has its proper place in laying the groundwork for this reassurance. Central sedation using the usual drugs, such as phenobarbital or sodium amytal, may be a useful adjunct when the patient is particularly unstable or very upset emotionally by prolonged worry concerning the significance of her complaints during the period before she sought medical advice.

2. *Physical Support*

The breasts, in these instances, are actually heavier than usual and need proper support to prevent the aching distress due to the dragging sensation produced by the traction of the pendulous organs on the pectoral fascia. Proper support does not mean merely suspending the breasts in tight brassières by narrow straps over the top of the shoulders, since this simply substitutes one complaint (shoulder distress) for the other (breast and pectoral discomfort). A correctly fitted brassière is essential in these cases and may need to be individually fashioned with a broad band around the lower chest and adequate strengthening of the lower part of the cup to support the weight of the breast above this fixed circumferential band without excessive traction across the shoulder top. Protection is, of course, also necessary at this period in order to avoid the pain of actual trauma to the tender, swollen breast and the brassière itself should be tight enough to prevent unnecessary motion of the affected breast.

3. *Physiological Support*

In this instance one attempts by various measures to overcome the local effect of the relative excess of œstrogen and the attendant stimulation of increased renal tubular reabsorption of sodium and water which produces the change in the extracellular fluid volume. These measures are most important during the 10 day period immediately before the onset of menstrual bleeding when the unopposed œstrogen effect is at its peak.

(a) *Use of diuretics.*—As noted above chlorothiazide (250 mg. to 500 mg. b.i.d.) and hydrochlorothiazide (50 mg. b.i.d.) have recently been used with striking success in the control of the excessive premenstrual weight gain so commonly encountered in these patients. This type of diuretic appears unequivocally the most effective yet discovered for this specific purpose. Dietary salt restriction during this period is, of course, an important contributory factor in the success of this therapy, but does not need to be as rigid as in the enforcement of this measure in cases of extreme myocardial insufficiency.

(b) *Use of vitamin B complex.*—Vitamin B_1 is considered to expedite the hepatic metabolism of circulating œstrogen but it merely assures that a normal process *can* take place. It does not really attack effectively the fact that there is a relative (and probably absolute) increase in the amount of œstrogen circulating in these cases. A dramatic response would not therefore be anticipated but occasional reports of symptomatic relief may encourage a trial of this therapy in cases not responding satisfactorily to the above measures. A daily level of 20 mg. to 25 mg. of thiamin hydrochloride in divided doses is considered an adequate amount when given, as is customary, during the latter half of each menstrual cycle.

(c) *Use of progesterone.*—Progesterone will not only help to overcome the renal tubular reabsorption of sodium and water thus diminishing the increase in extracellular fluid volume but it will also tend to produce a more normal sequence of changes from the proliferative to the secretory phase not only in the breast but also in the uterus. It might be expected consequently to improve the symptoms and signs of hyperplastic breast lesions as well as to provide relief from the pain associated

with non hyperplastic processes. Precautions in its use are necessary in order to protect ovulation, (if there is any indication that it is taking place) although most commonly it will be used only in those with anovulatory cycles.

Originally progesterone was available only for injection and this hampered its clinical application since daily administraton of 5 mg. to 10 mg. intramuscularly was considered the proper dosage level. However, oral preparations are now available and synthetic steroids have been prepared with progestational effects.

Both the oral preparations and the synthetic steroids are prepared by the manufacturer in 10 mg. doses and generally it is suggested that proper therapy should consist of one tablet once or twice daily from the midmenstrual or ovulatory period to approximately the 23rd to 25th day in the usual 28 day cycle, in order to permit progesterone-withdrawal bleeding to develop on schedule.

(d) *Use of androgens.*—Androgens are used with a view to producing pituitary depression of trophic stimulation of ovarian œstrogens and thus a diminution in their effect as the amount of endogenous œstrogen falls. Unfortunately, exogenous androgens are metabolized through a stage of biologically effective œstrogenic substances and these "exogenous" œstrogens are capable therefore of producing the same changes as the endogenous hormones so that the effectiveness of the therapy may not be apparent. Only occasionally is this type of treatment now considered worthwhile, but when all other measures fail, the use of linguets may in these rare instances provide the symptomatic relief required. It is customary to advise one linguet daily during the last half of the menstrual cycle as is true of the timing of the other measures described above.

(e) *Contraindications to œstrogen therapy.*—The use of œstrogen in symptomatic breast disease is clearly contraindicated. One might well digress to point out that careful consideration should be given to the use of œstrogens in the treatment of menopausal symptoms and the postmenopausal signs and symptoms of œstrogen deficiency. Hendrick[3] stresses the fact that since 1940, 60% of the patients in hi series having cystic disease or breast cance have had previous œstrogenic therapy. Thi is in keeping with the logical assumptio of the hazard of such treatment if these diseases are actually due to circulating œstrogen. Prolonged œstrogen administra tion has certainly produced mammary car cinoma in the male and is to be rigorously avoided in the high dosage here designed for pituitary depression. However, other feel that it has not, in limited dosage, pro duced any real increase in the incidence o breast tumours and that its benefit far outweighs its hazards.[16, 17] Nonetheless, the author does not feel that its use is warranted in these instances when other measures can be utilized.

Summary

The benign "physiological" breast diseases are *not* proven to be precancerous. Conservative management is, therefore, indicated. The normal and abnormal physiology of the breast (and uterus) are reviewed. The importance and frequency of the anovulatory cycle in producing symptomatic disease is noted.

The clinical syndromes appear to result from relative (or absolute) increase in œstrogenic stimulation of the breast. The benign breast diseases due to this factor fall into two groups; the non hyperplastic and the hyperplastic processes both of which are closely related to the syndrome of premenstrual tension since they all have in common the same basic ætiological relationships.

The principles of management of these benign "physiological" processes are outlined with a summary of one treatment programme designed to overcome and correct the basic fault responsible for the production of symptoms.

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RÉSUMÉ

Il n'est pas prouvé que les maladies bénignes "physiologiques" du sein soient pré-cancéreuses, d'où l'indication d'un traitement conservateur qu'on ne peut instituer que si on comprend la physiologie de base du sein et la physiopathologie que les maladies bénignes manifestent.

Les changements physiologiques au niveau du sein durant le cycle menstruel sont représentés dans le Figs. 1 et 2. Il est, en premier lieu, illogique de penser que les changements au niveau du sein seront des plus symétriques, cycle après cycle, durant toute la durée de l'activité génitale chez un même sujet. Il se produira des irrégularités et, selon leur localisation, des masses apparaîtront. En second lieu, les transformations mammaires ne surviennent que si l'hypophyse fonctionne normalement, et pour ce faire, nécessitent l'ajustement parfait de l'interrelation "hypophyse-ovaires". A cause de cette complexité, des aberrations peuvent être anticipées, et comme l'utérus et les glandes mammaires sont stimulés par les mêmes hormones, elles se rencontrent simultanément au niveau de ces deux organes, d'où l'association clinique fréquente des maladies mammaires avec les irrégularités menstruelles et la stérilité. Quoique la déficience en progestérone soit la cause déclenchante de l'inachèvement ou du dérèglement du cycle menstruel, pour toutes fins pratiques, c'est la concentration continue relativement élevée d'œstrone qui devient causale des maladies qui nous intéressent.

Dans la maladie non-hyperplasique (adénofibrose) les changements sont d'origine œstrogène indirecte, en tant que "stéroïd" produisant une augmentation de réabsorption de sodium (et d'eau) par les tubules du rein, avec accroissement du volume extracellulaire qui persiste dû à l'absence de diurèse compensatrice par excrétion du progestérone. Cette rétention se traduit cliniquement par le gonflement douloureux des seins et la réponse se manifeste par l'apparition de fibrose réactionnelle.

La maladie hyperplasique (mastite kystique chronique) d'autre part, résulte de l'effet direct local de la stimulation œstrogène sur le sein. Il se produit une prolifération anormale de l'épithélium et du tissu conjonctif péricanaliculaire. Le sein devient granuleux et nodulaire et l'accumulation de cellules autolysées dans les canaux est cause des écoulements mamelonnaires non physiologiques.

Le *support* constitue le point fondamental du traitement: le support psychologique afin de rétablir la stabilité émotionnelle, le support physique des seins pesants, enflés et douloureux et le support physiologique pour contrôler la rétention systématique et locale du à l'excédent œstrogène relatif. Sous ce rapport, les diurétiques sont employés pour diminuer l'excès du volume extracellulaire et dans les cas rebelles on peut tenter d'augmenter le métabolisme d'œstrone par la vitamine B_1 ou de contrebalancer son effet par addition de progestérone.

ADRENALECTOMY IN THE MANAGEMENT OF METASTATIC MAMMARY CARCINOMA: A FINAL EVALUATION OF 80 CASES

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A PRELIMINARY report[1] has already been presented concerning the results achieved by total œstrogen deprivation in the management of the end stages of disseminated breast cancer in a trial series of 32 patients. In this group all other methods of treatment had been tried previously and the major extirpation had been delayed until the metastatic disease was again active and progressive. No other potentially effective therapeutic tools were available. Consequently, the majority of the group so treated were in desperate straits with a limited life expectancy during which the patient faced grave and often totally disabling symptoms. The effectiveness of the procedure had to be assessed against this dreadful background.

It was in this connotation that Cade[2] referred to adrenalectomy as one of the most successful tools ever devised in the treatment of advanced malignant disease and in which sense Block *et al.*[3] have also more recently stated that "the endocrine ablative procedures have consistently given the highest percentage of success and the most profound remissions."

This type of enthusiasm has stimulated further interest in major extirpative therapy (including adrenalectomy and hypophysectomy) largely directed along the two important lines of study that are discussed below.

STAGE AT WHICH MAJOR EXTIRPATION SHOULD BE USED

In the first place the undoubted success of these operations when applied in the end-stage of the disease when the outlook is otherwise hopeless, quite logically begs the question concerning their application earlier in the course of the disease. Because cancer of the female breast is generally one which qualifies as a "good cancer" in the sense that the delicate balance between the invasive potential of the tumour and the resistance of the host is maintained in a relatively stationary and self imposed truce for long periods of time, it has been our policy to emphasize the importance of *sequential therapy* in these instances. This permits evaluation of the effectiveness of conservative measures which may offer some clinical information regarding the potential value of the major extirpation before one resorts to more serious therapeutic manœuvres of this type. The rationale of this philosophy of treatment in such instances has been reviewed in detail in previous publications.[4, 5]

In brief, it is thought that in these cases, in which the natural history of the disease is prolonged, factors inherent in the intrinsic growth and development of the tumour itself are of importance in determining this delay in tumour progression, in addition to the biological equation balancing biological aggressiveness of tumour against host resistance. In this sense the lethal, stem-line, or autonomous cell within the variegated tumour cell population is considered in competition with its siblings of dependent and less aggressive type for essential nutritive substances. If the lethal cell is in the minority quantitatively, it must exert all its energy for local survival when faced with this competition, and has no reserve with which to undertake invasion and destruction of host tissues. Sequential therapy envisages an attempt to maintain some semblance of this intratumour control by adversely affecting at any one time only a sufficient number of dependent cells to provide palliation while leaving others to continue the battle for survival with the autonomous cells in each local tumour conclave. It is thought that when applying therapeutic measures of this type in succession one after the other as the effectiveness of each individual procedure wears off, the cumulative longevity achieved will surpass that produced by an all-out attack on the sensitive components of the tumour population. In such a major attack one might anticipate a major destruction of the dependent cells leaving the potentially lethal cells now unopposed by any intratumour

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competition and, therefore, capable of transferring their energies previously expended of necessity for this purpose, into the pathways of host destruction. Since there has been no suggestion in any of the literature to date that hormonal alteration of the internal tumour environment can ever completely destroy these autonomous cells, one must always consider such treatment as a purely palliative gesture and adjudge its effectiveness in this connotation. The possibility of effecting "cure" of the disease in its metastatic phases does not appear pertinent in the present discussion.

An attempt is made in the accompanying figurative demonstration (Fig. 1 a, b, c, d and e) to illustrate these speculative considerations diagrammatically. While originally designed approximately a decade ago as a form of acceptable mental gymnastics in attempting to explain sequential therapy of this type when teaching the principles of cancer treatment, it is important to point out that objective support for these assumptions concerning the nature of tumour cell populations is now available.[6] The possible importance of variations in chromosome number in determining the biological nature of the tumour cell has also been reviewed recently.[4]

At any rate these originally hypothetical assumptions supported by such speculative mathematics as those reported by MacDonald and alluded to in previous publications[5] led us to continue reserving adrenalectomy, throughout the entire series under present review, for use at this late stage of the disease.

Nonetheless, it is only fair to digress for a moment to point out that breast cancer is not invariably a "good cancer" even in its early primary demonstration. It does occasionally exist as a "bad cancer" in which event the entire sequence of intratumour degradation of dependent cells, replacement by aggressive autonomous cells, invasion of the host by lethal cells, and eventual destruction of the host is accelerated enormously to produce an early fatal outcome. In these instances obviously an unvarying emphasis on sequential therapy would be ridiculous and the proper emphasis in treatment turns to one of more forceful application of any or all available measures.

The so called "acute" breast cancer is probably the most acceptable example of this type of "bad cancer" in mammary neoplasia. Treves[7] points out the lethal nature of this type of disease and the complete failure of radical local surgery to control the local growth or affect the eventual outcome. Stress is properly placed also on the fact that "acute" carcinoma does *not* include all cases demonstrating peau d'orange but is reserved for those with an erysipeloid reaction of the overlying skin in which redness and palpable marginal induration are the predominant features. Recent evidence[8] rather suggests that this reaction is fundamentally one of hypersensitivity thus implicating again the immune responses of the host now recognized of such importance in maintaining effective and prolonged control of tumour progression.[4] In appreciation of the hazard to the patient harbouring this specific lesion, and realizing the futility of accepted methods of managing the primary disease, Dao and McCarthy[9] report the results of total œstrogen deprivation when employed as the initial therapeutic gesture. In their group two or three cases of primary disease capable of evaluation showed prompt regression with one surviving 29 months without evidence of progression of the disease. They report also five patients with secondary inflammatory carcinoma treated in the same fashion, of whom three had an objective remission lasting in one case three and one-half years. No other method of treatment seems nearly as effective but since the number of cases available for comparison is so small, Fitts[10] quite correctly points out that before valid conclusions may be drawn, random sampling of a large series is essential. A co-operative effort by several centres with central statistical control seems clearly indicated if one is to answer properly this important question of determining what procedures might prove worthwhile for the patient with "bad cancer".

Type of Patient in Whom Major Extirpation may be Used

The present adrenalectomy series was

HYPOTHETICAL DIAGRAMS OF TUMOUR CELL POPULATION

Potential effect of graduated therapy

INITIAL TUMOUR CELL POPULATION

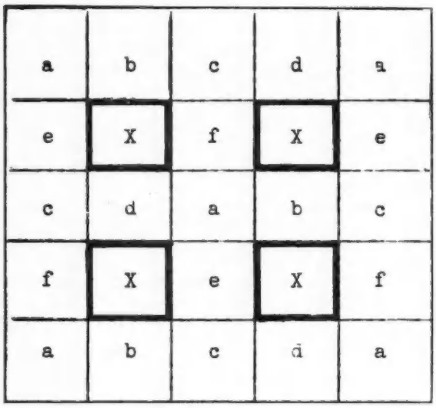


Fig. 1a.

$$\text{Ratio}\ \frac{\text{dependent cells}}{\text{autonomous cells}} = \frac{21}{4}$$

Each autonomous cell completely surrounded by dependent cells.

INITIAL THERAPY
(usually most effective)

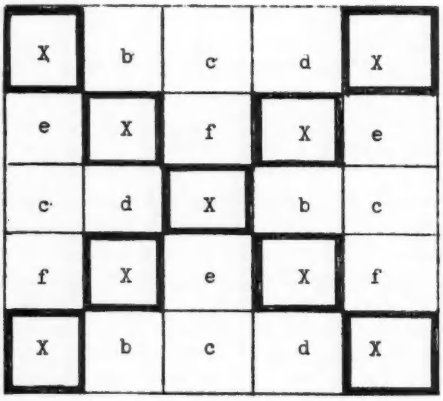


Fig. 1b.

$$\text{Ratio}\ \frac{\text{dependent cells}}{\text{autonomous cells}} = \frac{16}{9}$$

Each autonomous cell still has dependent cell neighbours.

ADDITIONAL THERAPY
(further destruction of dependent cells)

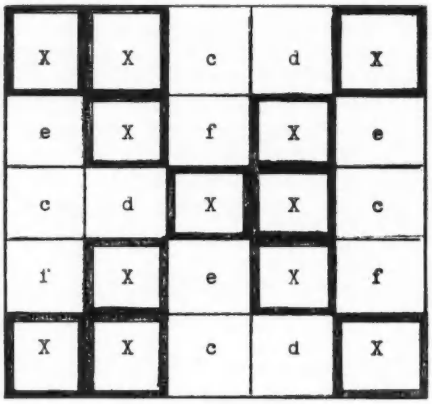


Fig. 1c.

$$\text{Ratio}\ \frac{\text{dependent cells}}{\text{autonomous cells}} = \frac{13}{12}$$

Clumps of autonomous cells beginning to appear.

FINAL THERAPY
(vigorous unaffected autonomous cells replace destroyed dependent cells)

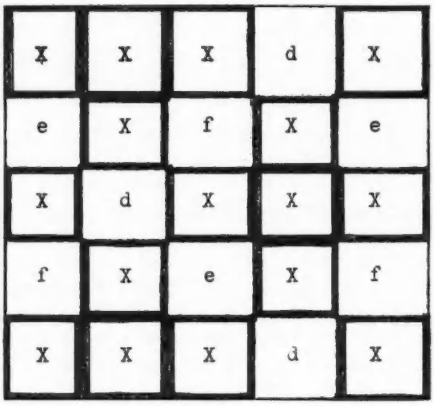


Fig. 1d.

$$\text{Ratio}\ \frac{\text{dependent cells}}{\text{autonomous cells}} = \frac{9}{16}$$

Balance now in favour of autonomous cells.

RAPID PROGRESSION
(despite treatment)

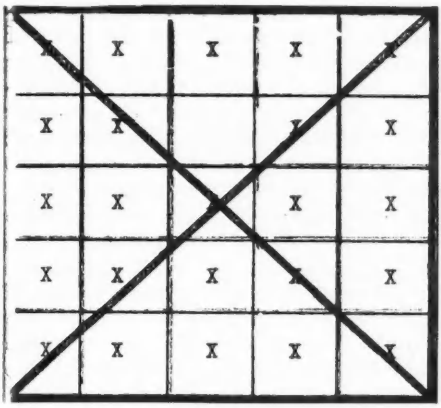


Fig. 1e.

Proliferation of fully autonomous lesion (capable of destroying own dependent cells).

Fig. 1a, 1b, 1c, 1d and 1e.—As a result of the initial therapy the most sensitive (and most numerous cells) (type "a") are destroyed. Hence there is appreciable regression of tumour mass and resultant improvement in symptoms and signs until eventual replacement of these cells by the autonomous group (type "X").

Additional therapy may affect other sensitive cells (type "b") to produce remission. Subsequently replacement by autonomous cells results in clumping of the type "X" cells into colonies for more effective proliferation.

The final therapy affecting the majority of the remaining dependent cells (type "C") results, on replacement proliferation of the autonomous cells, in a tumour population now heavily weighted in favour of these aggressive "X" cells.

Rapidly thereafter any persisting sensitive cells (types "d", "e" and "f") may disappear by the process of biological and physiological degradation to leave a fully autonomous and, therefore, lethal tumour.

KEY

X = autonomous tumour cell

a, b, c, d, e, f = dependent tumour cell

cf. TOTAL THERAPY
Combined use of many or all available methods of treatment.

If successful in eradication of all dependent cells may leave autonomous cells without restraint of competition with other types of tumour cells. It may therefore have stimulated the early appearance of an aggressive lethal tumour.

established in three stages. A preliminary study of adrenalectomy and oophorectomy in 32 cases previously reported[1] was evaluated in an attempt to create clinical indications for the procedure. The second stage involved an additional 48 patients in which total œstrogen deprivation of this type was performed keeping in mind the indications previously developed. Quite naturally the indications were not made absolute since the evidence was not in any way conclusive. Nonetheless, the incidence of definite objective improvement rose from 40.6% in the initial series to 50% in the entire group under assessment when these basic fundamental policies were recognized. The third stage, to be completed before publication of the final evaluation of the procedure, included a follow-up of the patients until they eventually succumbed to the disease, in order that firm figures for length of objective remission and total survival could be determined. Consequently, after completing the treatment of these 80 cases the study was terminated until the present report could be prepared.

It is encouraging to note how well the clinical indications originally established[1] have stood the test of time. On physiological grounds it is, of course, clearly recognized that ovarian function must be controlled when adrenalectomy is performed in order to prevent subsequent trophic stimulation of ovarian œstrogens.[5] In general it is accepted that castration is indicated up to the age of 60 at least. A prior castration followed by objective remission is an indication that further œstrogen deprivation by adrenalectomy may be expected to provide another period of palliative relief.

The clinical considerations suggested earlier to be of importance might be sum-

marized as follows:—

1. A previous trial of all other available methods with uncontrolled disease at the time of considering adrenalectomy.

2. A long history of disease from the initial recognition of the primary lesion to the presenting situation.

3. A favourable response to previously exhibited therapy indicating a delicate balance between the tumour activity and the host resistance, which can be easily tilted in favour of the host.

4. A remission after castration was considered a particularly favourable feature.

5. The presence of metastases principally involving the skeletal or local soft tissues, with the vital organs (and particularly the liver and the central nervous system) free of clinical disease.

6. The occurrence of such end-stage disease in a person considered to be a psychologically suitable candidate for additional major surgery.

Of particular interest in this regard is the recent report by the Ann Arbour Group[3] which in essence presents the same conclusions. They stress, as we have done in the past,[5] that careful restriction in the use of adrenalectomy is mandatory in view of the adverse effect of the procedure if the patient does not show a marked objective remission. They suggest its use only if there is no evidence of liver involvement, if there has been a previous response to oophorectomy, and if there is a prolonged duration of the disease (over 3 years) with the patient preferably in the age group between 50 and 60 years. As an additional feature of importance the prognostic significance of high urinary œstrogen levels is noted.

In this latter regard the value of laboratory investigations to date has proved limited and often disappointing. The œstrogen stimulation and the cortisone inhibition tests[11] have not been widely applied because the lack of response did not prove to be an absolute indication of tumour autonomy and the hazard of hypercalcæmia in patients with dependent skeletal lesions is considered to be very real. The biological methods of assay of urinary œstrogens are unreliable, sometimes even in skilled hands, and consequently chemical techniques of measuring urinary œstrogens are necessary. Block *et al.*[3] have turned to paper chromatography and suggest that success may be achieved in roughly two-thirds to three-quarters of the patients if operation is restricted to those who excrete relatively large amounts of urinary œstrogen. Palmer and Helstrom,[12] on the other hand, have used chemical methods without determining any clear-cut indication of the result to be anticipated. Vaginal smears with cornification indicating the presence of biologically effective œstrogens have similarly not proven universally acceptable. Despite the need for a definitive test of prognostic value we are still faced with the problem of making the decision to utilize major extirpative therapy largely on the clinical grounds previously outlined.

Results of Total Œstrogen Deprivation:

(*Adrenalectomy Alone or with Oophorectomy*)

The present survey follows the patients on whom these procedures were performed (Table I) up to the end of 1958 so that no patient was followed-up for less than one year. Only three patients were alive at this time (49 months, 46 months, 42 months) although one had demonstrated recurrent disease. It was felt that their inclusion in a final report would not significantly affect the accuracy of the average remission and survival periods quoted below.

The same indications pertain in the selection of cases for treatment as were outlined in the preliminary report, and identical conclusions were drawn as to the type of disease most likely to be helped in a worthwhile manner. A prolonged period between the recognition and treatment of the primary lesion and the consideration of adrenalectomy represented the most favourable situation, although in two cases it was found that success with previous therapy

TABLE I.—Incidence of Adrenalectomy

Year	*Number of cases*
1953	19
1954	31
1955	17
1956	7
1957	6
Total	80

had created a completely autonomous lesion which failed to respond to the operation as had been anticipated. In no instance did we observe frank exacerbation of the lesions as had been suggested might theoretically occur were the mammotrophic complex to be released by the ablation of adrenocortical inhibition of the pituitary. It is quite possible that this is due to the policy followed in this series of restricting the major extirpations to end-stage disease in which event exacerbation, if it did occur, would scarcely be detectable clinically since worsening of the patient's condition might well not be apparent.

In this series a strictly clinical preoperative evaluation was utilized in the selection of suitable patients. It was not possible to evaluate the usefulness of calcium excretion studies, the stilbœstrol stimulation test, or the cortisone inhibition test, nor were endocrine assays used with measurement of gonadotrophins or œstrogens. The possible value in prognosis of estimating steroid metabolites has previously been mentioned and undoubtedly all these methods may be useful in isolated cases in choosing suitable candidates for major extirpative therapy. However, the proven ability of the host to contain the neoplasm remains the single most important factor influencing clinical judgment concerning this procedure.

1. *Mortality and Morbidity Statistics*

The accompanying tabulations summarize the experience developed in this group of patients. The mortality (Table II) and morbidity (Table III) attendant upon a major operation of this type in such ill patients is not excessive and most of the deaths resulted from steady progression of the unaffected disease. No operative catastrophes are recorded but several of the early postoperative deaths might be attributed to the additional insult of the operation and the anæsthetic. As is customary, deaths up to the end of the first postoperative month have been included in the mortality rate, but in these late fatalities the end result could scarcely be considered a complication of the surgical procedure, except in the single case in which we were unable to control a persistent sodium deficiency. Perhaps with the 2-methyl 9-α-fluoro corticoids now available this catastrophe might have been avoided, although it occurred in a patient failing to show other evidences of a successful result. Two of these patients were particularly unfortunate as they both had dramatic sub-

TABLE II.—Postoperative Mortality

Within one month

1st day—respiratory insufficiency—extensive pulmonary metastases.
2nd day—respiratory insufficiency—extensive pulmonary metastases.
10th day—sudden death—? collapse vital centres.
10th day—progression of disease.
11th day—cardiovascular collapse: dyspnœa: supraventricular tachycardia.
13th day—perforated duodenal ulcer (unrecognized before autopsy).
14th day—cerebral anoxia with coma (following first stage of two-stage adrenalectomy with oophorectomy).
16th day—cardiovascular collapse—malignant pleural effusion, gross hepatomegaly (metastatic).

At end of first month

—Progressive cerebral metastasis.
—Progression of disease complicated by postoperative parotitis and hæmaturia.
—Persistent Addisonian state not controlled.
—Recurrent hypotensive crises (myocardial disease). BUT dramatic relief of pain.
—Spontaneous paradox of thoracic cage (pathological fracture) BUT dramatic relief of pain.

TOTAL 13 deaths in 80 cases: mortality rate of 16%.

TABLE III.—Postoperative Morbidity
(within 1 month)

1 Wound dehiscense—stitches removed in error on third postoperative day.
1 Tension pneumothorax at 48 hours (treatment: aspiration).
1 Postoperative parotitis (treatment: radiotherapy) with hæmaturia (treatment: suprapubic cystotomy).

Postoperative Complications
(after 1 month)

1 Addisonian with extensive skin pigmentation.
2 Recurrent Addisonian crises.
1 Sensitive to cortisone. Adequately maintained on hydrocortisone.
1 Died of unrecognized tension pneumothorax following radioactive collodial gold injection (operation ineffective).
1 Disease reactivated by accidental injury to bony metastases at 6 months. (Excellent remission following operation).
1 Died of cardiac infarct at five months. (Excellent remission following operation).
1 Suicide at five months (lævo-dromoran). (Operation ineffective).

jective relief of osseous pain only to succumb in one instance to myocardial disease and in the other to a mechanical problem associated with spontaneous pathological rib fractures and instability of the chest wall.

The morbidity within the first postoperative month, and the complications encountered after the first month are unimpressive since a direct causal relationship between the adrenalectomy and serious complaints could not be firmly established (Table III). The Addisonian patients might well be adequately controlled by the newer synthetic steroids, although it is interesting to record that failure to control the underlying disease was an invariable accompaniment of this problem.

2. *Postoperative Survival*

The present report deals primarily with the actual results obtained and therefore, the period of remission and the length of survival are the two features in which we were most interested.

The survival periods in the 67 patients surviving the operation are enumerated in Table IV, each entry noting the closest month at which death resulted. Only three patients had any additional treatment, in each instance a hypophysectomy from which no apparent additional benefit was detected, and it was not thought that the length of survival was prolonged in these instances. Three of the group are still alive, all at approximately the four year period, although one of these now shows evidence of reactivation of the disease. It is interesting to note that nearly 40% of the patients lived longer than one year from the date of the operation despite their desperate state immediately before the surgical procedure. The general picture of the survival pattern is summarized at the foot of Table IV.

The average survival times are next compared in Table V dependent upon the type of clinical response to the procedure. Subjective remissions include relief of pain, dramatic improvement in the patient's sense of well being, control of dyspnœa etc., whereas objective improvement implies some measurable alteration in the size of

TABLE IV.—Survival Period after Œstrogen Deprivation

Adrenalectomy ..... 40 cases
Combined adrenalectomy and oophorectomy ..... 27 cases

First year months	*Number*	*Following adrenalectomy*	*Following combined adrenalectomy and oophorectomy*
1	2	2	0
2	7	4*	3
3	2	1	1
4	2	1	1
5	2	1	1
6	4	4	0
7	5	4*	1
8	2	0	2
9	4	3	1
10	3	2	1
11	2	0	2
12	1	1	0
Totals	36	23	13
Second year months			
13 - 15	6	2	4
16 - 18	5	3	2
19 - 21	4	3*	1
22 - 24	4	2	2†
Totals	19	10	9
Third year months			
25 - 27	5	4‡	1
28 - 30	3	1	2
31 - 33	0		
34 - 36	0		
Totals	8	5	3
Fourth year months			
37 - 39			
40 - 42	2		2
43 - 45			
46 - 48	2 (1 at 49 months)	2	
Totals	4	2	2

*Previous surgical oophorectomy in one patient of each group (three patients).

†1 patient had hypophysectomy two months before death.

‡2 patients had hypophysectomy five months before death.

SUMMARY:—36 patients died in the first year.

BUT
31 lived longer than one year.
(38.75% of entire group of 80 cases).
20 lived longer than one and one-half years.
12 lived longer than two years.
4 lived longer than three years.
1 lived longer than four years.

AND
3 patients are still alive.
one at 49 months
one at 46 months (with disease)
one at 42 months.

TABLE V.—Comparison of Average Survival Times After Œstrogen Deprivation

Clinical response	Total group (80 patients)			Series exclusive of postoperative deaths (67 patients)			
	Number of patients	Percentage	Average survival time in months	Adrenalectomy (40 patients)		Combined adrenalectomy and oophorectomy (27 patients)	
Postoperative mortality	13	16.25	<1	Number of patients	Average survival time	Number of patients	Average survival time
No subjective or objective remission...	15	18.75	4.1	7	months 4.1	8	months 4.1
Subjective remission but no objective change..	12	15	8.7	11	8.6	1	10
Both subjective and objective remission...	40	50	19.5	22	19.0	18	20.1
Totals.....	80	100	Exclusive of postoperative deaths 14.1 months (67 patients)	40	13.7	27	15

palpable disease or in radiologically detectable pulmonary metastasis, radiological healing of osteolytic disease or resorption of effusions. If no definite evidence of this type could be discovered despite the clinical impression of a favourable response the patient was considered to show subjective improvement only. In comparing adrenalectomy, and adrenalectomy plus oophorectomy, one is impressed that the comparative figures for the two procedures utilized are so similar, implying that the actual degree of œstrogen deprivation provided in each instance is also similar. As in the previously reported series adrenalectomy alone was reserved for those patients who were at least five years beyond the last evidence of menopausal symptoms of any type. Recent evidence[13] that ovarian activity persists for longer periods than this, might suggest that the oophorectomy should also be an integral part of the programme until the age of at least 60 years.

Those patients who demonstrated no improvement at all showed identical survival periods of 4.1 months with seven patients in one group and eight in the other. Only in the portion of the entire group who showed subjective but no objective remission is the series unbalanced, with a preponderance of the 11 cases undergoing adrenalectomy and only one having also a bilateral oophorectomy. However, the survival periods did seem similar with a definite improvement noted when compared to those who failed to respond in any way, suggesting that the extirpation did have a favourable (although limited) influence on the pattern of survival.

A striking increase in the length of survival is apparent as might be anticipated in those who showed definite objective signs of regression of the metastatic disease. Here again the series is fortuitously well constructed with 22 patients in one group and 18 in the other and as in the other comparisons the total survivals were almost identical averaging 19.5 months. It is worth noting that this group represented 50% of the entire series indicating that an appreciable degree of œstrogen dependence persists in many of these cases despite all the various methods that had been utilized in the management of the problem previously. In none of the patients in this series was there any evidence that the œstrogen deprivation resulted in exacerbation of the disease, as does occasionally occur in additive hormonal therapy. Isolated instances have been reported following adrenalectomy and oophorectomy in which exacerbation did occur presumably due to the release of the mammotrophic complex (prolactin and/or growth hormone) but this complication has not been recognized in this series.

The fact that those showing objective improvement only rose from 40% in the initial trial series to 50% in the entire group, indicates the difficulty in selecting

TABLE VI.—Comparison of Duration of Remission and Survival Time

Total Group

(exclusive of postoperative deaths)

Clinical response	*Number*	*Average remission (months)*	*Average survival (months)*	*Survival after reactivation (months)*
No subjective or objective remission	15	0.0	4.1	4.1
Subjective remission but no objective change	12	2.75	8.75	6.0
Both subjective and objective remission	40	14.0	19.5	5.5
Totals	67	8.8	14.1	5.3
Adrenalectomy (40 patients)				
No subjective or objective remission	7	0.0	4.1	4.1
Subjective remission but no objective change	11	2.7	8.6	5.9
Both subjective and objective remission	22	15.1	19.0	3.9
Totals	40	9.0	13.7	4.7
Combined adrenalectomy and oophorectomy (27 patients)				
No subjective or objective remission	8	0.0	4.1	4.1
Subjective remission but no objective change	1	3.0	10.0	7.0
Both subjective and objective remission	18	12.7	20.1	7.4
Totals	27	8.6	15.0	6.4

favourable cases on clinical grounds alone, although the same general indications were still apparent in the final analysis. Continuing attempts to evaluate laboratory means of demonstrating persistence of œstrogen dependence are, therefore, essential in trying to make certain that a major extirpative procedure of this type is offered only to those for whom a reasonable expectation of success is held. Certainly the patient who fails to respond is made worse by these procedures in the sense that she is then less able to react favourably to intercurrent stresses as well as being less capable of managing the terminal stages of the disease itself.

3. *Postoperative Remission*

Prolongation of life alone, of course, cannot be considered worthwhile palliation even if associated with objective evidence of regression of the disease, unless relief of suffering is also associated with the favourable response.

In Table VI an attempt is made to distinguish the length of remission during which the patient remained relatively asymptomatic and also the period of survival after symptomatic (or objective) reactivation of the disease. In those with subjective improvement only, the close similarity persists in the pattern noted above, with palliation averaging 2.7 months and survival after reactivation six months. When objective changes were detected, palliation was effective for an average of 14 months, again a striking improvement of similar degree in both methods of treatment under comparison. It is interesting to note that the survival after reactivation however, even in this favourable group, remains only 5.5 months and closely resembles the period of six months, when only subjective improvement occurred, and also the survival of 4.1 months when the operation was ineffective. These figures presumably reflect the results of the tumour-host conflict when an autonomous tumour has been created, partly at least as the result of the previous treatment to which it has been subjected.

Preliminary Assessment of "Medical Adrenalectomy"

Despite the encouraging results achieved in this series and similar reports from other sources, the disadvantages attendant upon an ineffective adrenalectomy have led to attempts to duplicate its effect by non operative and potentially reversible means. The rationale of such an approach has been thoroughly reviewed in a companion article[5] and will not be discussed here apart from stressing the fact that cortisone inhibition of pituitary stimulation of adrenal œstrogens can, as in the case of surgical adrenalectomy, be successful only if the resultant increase in gonadotrophic stimulation of ovarian œstrogen is prevented by ablation of any functioning ovarian tissue.

During the third phase of the currently reported study, while awaiting the final outcome in the surviving patients, a preliminary appraisal of the effectiveness of cortisone therapy in similar situations was made. The results are recorded in Table VII and as might be anticipated when considering the nature of the more definitive and positive surgical procedure, they are less dramatic than those achieved by surgical adrenal-

TABLE VII.—Average Remission and Survival Periods With Cortisone Therapy
(Preliminary report of initial 29 cases)

Clinical response	No. of patients	Percentage	cf adrenalectomy	Average remission	cf adrenalectomy	Average survival	cf adrenalectomy
No subjective or objective remission	8	28	35 (includes postoperative mortality)	0	0	< 2 mos.	2.6 mos. (includes postoperative mortality)
Questionable subjective remission but no objective change	5	17 (45)		—		3 mos. (5 mos.)	
Subjective remission but no objective change	3	10	15	1 2/3 mos.	2.75 mos.	3 1/3 mos.	8.7 mos.
Both subjective and objective remission	13	45	50	7 mos. (3 still in remission)*	14.0 mos.	9½ mos. (7 still alive)†	19.5 mos.
Totals	29	100	100	*Best result—24 mos. remission †Best result—30 mos. survival			

ectomy where average remissions and average survival times are approximately twice as lengthy as in "medical adrenalectomy". The percentage of patients affected favourably, however, is approximately the same, indicating the basic similarity of the procedures and the similar relative success to be anticipated if the same clinical situations pertain.

Lemon[14] has recently reviewed in scholarly fashion the place of cortisone and prednisone in the treatment of advanced mammary cancer. The effectiveness of these hormones may admittedly be predicated not only on their ability to produce adrenocortical suppression but also on a direct antineoplastic growth effect due to alterations of the internal cellular environment as suggested by the evidence to be quoted here.

In the first place, there is unquestionably a high incidence of ovarian stromal hyperplasia in patients with breast cancer, and even in postmenopausal women harbouring the disease the amount of œstrogenic substances in the urine may approximate the quantities recorded in normal women without breast cancer and still in the years of active sexual life. Corticoids have been demonstrated to be antagonistic on the tissue cell level to the effects of œstrogen and may be considered as œstrogen antagonists.[15] Progesterone and androgenic steroids are also physiologically antagonistic to œstrogen but these substances rapidly disappear after the menopause, and the corticoids remain as the only effective antagonists to the action of œstrogen on mammary tissue.

Following this line of thought Lemon[14] also reports a decrease in corticoid metabolites in untreated active metastatic breast cancer, and a decreased ratio of corticoid to œstrogen secretion may well represent the type of environment necessary to the continuing activity of mammary neoplasms.

Allen, Hayward, and Merivale[16] similarly report a higher ratio of 11desoxy 17-ketosteroid metabolites to 11oxy ketosteroids in treatment success after œstrogen deprivation than in treatment failures, suggesting that benefit occurred in those with active sex hormone production and consequently an increase in the œstrogen to corticoid ratio. This ratio could presumably be reversed by treatment. Hence one of the basic reasons for the success of corticoid therapy may be simply represented by the reversal of this trend and the creation of an environment not so favourable to the tumour. Accurate biochemical estimations of this kind may subsequently provide more certain information on which to base an intelligent prognosis as to the possible effectiveness of cortisone therapy, and therefore a more definite indication for its use than exists in the highly theoretical atmosphere of hormone therapy at the present time. The single fact that a favourable response has been reported when cortisone has been used in the treatment of remissions following major extirpative therapy, also indicates the potentiality of this additional effect.[17]

The further observation that cortisone may be peculiarly effective in the treatment of hypercalcæmia and in the palliative management of metastatic hepatic disease with relief of obstructive symptoms

as a result of the direct anti-inflammatory action of glucocorticoids, should not be ignored.[18] The euphoria occasionally produced although not indicative of a favourable objective response, is nonetheless a welcome occurrence in the unfortunate, anxious and depressed patient facing the challenge presented by the end-stage of this disease.

Discussion

These results stimulate thoughtful comparison of the effect of the two methods of controlling adrenocortical œstrogen production. In retrospect it would appear that the determining factor in considering surgical adrenalectomy is the evidence that 50% of the patients, despite attempts at clinical differentiation of favourable cases, do *not* demonstrate remission in their disease, in which case the burden of Addisonian deficiency is added to the heavy load already imposed on patients by their disseminated disease. Since this change is irreversible the risk of such an eventuality may well be prohibitive.

This is particularly true since the survival period is limited (average 19.5 months) and the period of actual remission averages only 14 months even in the most favourable regressions. Since there are no curative implications here, the dividends from this therapy may not be worth the gamble inherent at present in the operation. This conservative position is fortified by the observation that cortisone inhibition of adrenal œstrogens affects favourably the same group of patients as responded to surgical adrenalectomy, albeit less dramatically. Most would feel, we are sure, that the difference in remission and survival periods averaging at best less than seven months and 10 months respectively, is scarcely adequate reason for substituting an irreversible procedure with a 50% failure rate, for one which is reversible if unsuccessful.

Nonetheless, the dramatic response recorded when effective, makes it mandatory that continuing attempts be made to predict the response to adrenalectomy more accurately. If the high urinary œstrogen excretions measured chemically did actually prove to permit utilization of adrenalectomy with a chance of success varying between 66% and 75% as suggested by Block *et al.*[3] then, of course, this emphasis would be reversed and adrenalectomy would become a very attractive concept.

In utilizing adrenalectomy in a sequential programme of therapy the procedure should be deliberately reserved for a last ditch stand in the treatment of the usual "good cancer" but one must admit that it becomes a matter of delicate clinical judgment to decide the exact point at which is should be used because of its ineffectiveness when vital organs, such as the liver and the central nervous system are seriously affected. Consequently, it becomes a tool to consider as soon as the established methods of treatment have been proven to be ineffective, and these methods should not be continued, unless they are producing valuable objective responses, simply for the sake of doing something for the patient.

Established methods of treatment whether surgical, radiotherapeutic or hormonal in type are considered to be already ineffective when the lesion under treatment presents as an "acute" carcinoma. In such instances of "bad cancer" the major extirpations are perhaps most clearly indicated since an all-out attack on the disease is now necessary if any worthwhile results are to accrue from treatment. The principle of sequential therapy is not to be considered in these specific instances.

A comparison of surgical adrenalectomy and hypophysectomy would be invidious in the type of presentation here recorded. The problem has been reviewed previously.[5] Much of the comment concerning adrenalectomy outlined above is directly applicable to any consideration of hypophysectomy in this disease.

Summary

A series of 80 cases having surgical adrenalectomy as part of a programme of total œstrogen deprivation has been studied to the final fatal outcome in each patient. The stage of the disease at which major extirpative therapy becomes indicated is discussed. This falls largely within the scope of sequential therapy in most breast cancers. Only in the so-called

"acute" carcinoma is a bold attack, including adrenalectomy indicated as a possible initial therapeutic gesture.

The situational syndromes in which major extirpations may be considered are reviewed. Clinical and laboratory findings are evaluated in this regard.

The results achieved by total œstrogen deprivation are summarized in tabular form. Approximately 50% of patients accepting surgical adrenalectomy in the end-stage of their disease will demonstrate an objective remission averaging 14 months in duration, and a postoperative survival averaging 19.5 months.

A preliminary assessment of "medical adrenalectomy" is studied in comparison to the results of adrenalectomy.

It is felt that the irreversible nature of a major surgical extirpation and the fact that 50% will *not* demonstrate a remission, gives cortisone inhibition of the adrenal a preferable emphasis.

Adrenalectomy should only be considered in the most favourable instances with the same indications previously reported[1] still representing the best basis for clinical assessment of the problem. A prolonged history, previous response to castration and lack of any evidence of involvement of vital organs are fundamental requisites. Since successful adrenalectomy still represents the most effective tool yet available in the management of metastatic mammary disease, continued emphasis on means of predicting such a response is mandatory. If available, the indications for the operation might well be extended.

ACKNOWLEDGMENT

Much of the success of this study is directly due to the financial backing of the Ontario Cancer Treatment and Research Foundation who supplied the monies necessary to support the replacement postoperative cortisone therapy in these patients, free of charge. This help is very deeply appreciated.

The author also wishes to thank particularly, Dr. C. L. Ash and Dr. M. Vera Peters of the Ontario Cancer Institute at the Toronto General Hospital, whose unfailing interest added the major source of patient material for development of the study.

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RÉSUMÉ

Il est encourageant de noter combien les indications cliniques déjà établies[1] ont soutenu l'épreuve du temps. Physiologiquement parlant, il

est admis que la fonction ovarienne doit être contrôlée lorsque la surrénalectomie est pratiquée, afin de prévenir toute stimulation trophique subséquente d'œstrone ovarienne.

On admet, en général, que la castration soit indiquée jusqu'à 60 ans au moins. Une castration antérieure suivie de rémission objective, constitue une indication que la privation poursuivie d'œstrone par surrénalectomie devra amener une autre étape palliative.

Cette présente étude, qui vise surtout à montrer des résultats, nous intéresse donc du point de vue rémission et survie, ce qui est exposé aux graphiques 4 et 5.

Il est intéressant de noter que près de 40% des patients ont survécu plus d'un an, malgré leur état pré-opératoire désespéré. Les rémissions subjectives comprennent le soulagement de la douleur, l'amélioration dramatique de l'état de santé du maladé, le contrôle de la dyspnée, etc., alors que les signes objectifs d'amélioration comprennent le changement de volume des masses palpables ou des métastases pulmonaires radiologiquement décelables, la guérison radiologique des lésions ostéolytiques ou la résorption des épanchements.

Alors que cette étude se poursuivait, une appréciation préliminaire de l'éfficacité d'un traitement avec la cortisone dans des situations identiques était entreprise et, des résultats exposés au graphique 7, il ressort que la "surrénalectomie médicale" semble être de 50% inférieure à l'opération.

L'observation menée plus loin, cependant, qui montre que la cortisone soit étrangement effective pour traiter l'hypercalcémie et amener une palliation du côté des métastases hépatiques, avec amoindrissement des symptômes d'obstruction, ne devrait guère être ignorée. De même, l'euphorie produite occasionnellement, ne peut être que bienvenue pour ces malades anxieux et déprimés.

Il ressort donc qu'à cause de l'irréversibilité de l'acte chirurgical, et de l'absence de rémission dans 50% des cas, l'inhibition surrénalienne par la cortisone soit préférable.

La surrénalectomie ne devrait être considérée que pour les cas dont l'évolution est de longue durée, qui ont déjà répondu favorablement à la castration et qui ne présentent aucune évidence de métastase aux organes intéressés.

FUNDAMENTAL TECHNIQUES OF PLASTIC SURGERY AND THEIR SURGICAL APPLICATIONS. Ian A. McGregor, M.B., F.R.C.S. (Eng.), F.R.F.P.S.(Glas.), Consultant Plastic Surgeon, Glasgow Royal Infirmary. Foreword by C. F. W. Illingworth, C.B.E., M.D., Regius Professor of Surgery, University of Glasgow. 244 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London. The Macmillan Company of Canada Limited, Toronto, 1960. $5.00.

This is a small, well written, well illustrated book that seems aimed at being a liaison piece between the general surgeon and the plastic surgeon. This it does, and both will find common ground. The fundamental principles and rationale of wound care, local tissue shifts, flaps and pedicles are there for the general surgeon. They will also help students in plastic surgery and even the senior plastic surgeon can use the book to polish up his thinking and refurbish his teaching diagrams of procedures he has long taken for granted.

It is not an all inclusive textbook on plastic surgery and contains nothing on the treatment of face fractures, cleft lip and palate, rhinoplasty and otoplasty and other things which are fundamental parts of plastic surgery.

The first part describes the basic techniques of plastic surgery in detail. Wound care, incision placement, stitchcraft, the Z-plasty, varieties of free skin grafts, flaps, pedicles and tubes are described in logical, well illustrated sequence. The woefully sketchy second part considers the applications of these principles to general surgery and other specialties.

This is a good basic textbook and may well take its place with the fundamental texts of other specialties. The format and printing are pleasant, the illustrations are clear and well chosen and the price is most acceptable.

SURGICAL TREATMENT OF BONE AND JOINT TUBERCULOSIS. Robert Roaf, Department of Orthopædic Surgery, Liverpool, W. H. Kirkaldy-Willis, Orthopædic Centre, Nairobi, Kenya., and A. J. M. Cathro, Orthopædic Centre, Nairobi. 137 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London., The Macmillan Company of Canada Limited, Toronto, 1959. $5.00.

This volume represents the present day approach to the management of bone and joint tuberculosis. With the availability of p-aminosalicylic acid (PAS), isoniazid and streptomycin, the surgeon has become courageous in his direct attack on this disease. The authors emphasize this point using chemotherapy as a protective cover. The techniques of debridement or combined debridement and fusion, are discussed with enthusiasm and confidence.

The book is well organized, dealing with each area separately. Chapters on the spine and Pott's paraplegia are particularly well done and touch on the anterior approach to the vertebral column, indicating the authors' satisfaction with this approach. The size of the book does not allow for the details of operative technique in these problems.

Chapter after chapter reveals the direct surgical attack on the tuberculous lesion to be the most satisfactory in all but the earliest cases. The illustrations are clear and adequate in amount.

The authors are to be congratulated on this work. It is well written, concise and clear and is to be recommended to the orthopædic surgeon who desires a dynamic description of the surgical treatment of bone and joint tuberculosis.

SIGNIFICANCE OF CLOSTRIDIUM WELCHII INFECTIONS AND THEIR RELATIONSHIP TO GAS GANGRENE*

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OFTEN THE REPORT on a wound swab "*Cl. welchii* present" starts a sort of witch hunt in a hospital. The patient is strictly isolated, the operating room is scrubbed time and again and perhaps left vacant for 24 to 48 hours, and crackling messages go from medical superintendent to floor nurses, to senior surgeon, to infection control officer, to bacteriologist and to operating room supervisors, but not necessarily in that order. Can this attitude be justified? The question will be considered in this paper which reviews all cases of *Clostridium welchii* infection in a large hospital over a five year period, from 1955 to 1959, and speculates on their relationship to gas gangrene.

Bacteriologists often see this organism in routine smears from wounds, and tend to deprecate its significance in this hospital, not reporting it unless present in appreciable numbers, or if it appears significant from the case history. *Clostridium welchii* (Syn./- *Cl. perfringens*, and in older literature, *Bacillus aerogenes capsulatus* or *B. welchii*) is readily recognized and identified. In smears it is recognized morphologically as a rather large, thick, Gram-positive rod. In anaerobic culture, its colonies can be identified fairly easily. *However, bacteriologically, it is impossible to differentiate an organism causing true gas gangrene from a simple contaminant.* To quote MacLennan,[1] "Anaerobic infections are not bacteriological but clinical entities; this has been frequently emphasized but not widely appreciated. The same pathogenic anaerobe can cause widely different conditions; thus *Cl. welchii* may equally well be found in a healthy wound, and in a man dying from gas gangrene."

It is known that organisms other than *Cl. welchii* may produce gas gangrene, but in a large series of such infections, 80% were caused by *Cl. welchii*, 10% by *Cl. septique*, and 3% by *Cl. œdematiens*.[2] For practical purposes, it may be accepted that nearly all cases of gas gangrene in civilian hospitals are caused by *Cl. welchii*.

Clostridium welchii is widespread in nature, being present in soil, dust, sewage, milk, water, woollen clothing, the fur of domestic animals, and in the *normal* gastrointestinal tract of man and animals. Since the organism is so common, it is a wonder that gas gangrene is relatively rare, although as many as 10% of serious wounds were said to be complicated by gas gangrene in the early part of World War I. In World War II the incidence was decidedly lower: in a large series of 187,936 major open wounds, gas gangrene occurred in only 1.76%.[2] The bacterial flora of open wounds is often similar in those that develop gas gangrene and in those that do not. Although dead material in wounds, especially dead muscle, and interference with the blood supply, favours the onset of gas gangrene, the problem still exists that of 100 similar wounds perhaps two will develop this dread infection. One can only infer from this that some strains of *Cl. welchii* are more virulent than others; but *which strains are most virulent* is still an unanswered question. A city may have no cases of gas gangrene for several years, and then three or four in a one or two month period.

Clinically, it has been recognized that *Cl. welchii* in a wound may:—(1) remain as a simple contaminant, (2) cause an anaerobic cellulitis, or (3) cause true gas gangrene (Clostridial myositis).

Over the years, clinicians have become aware of another clinical entity, fairly common in civilian practice following major surgery, that of *Cl. welchii* wound infection, or colloquially "Welch abscess". This is perhaps an end result of anaerobic cellulitis, and one may be splitting hairs by mentioning it, but the fact remains that in my experience "cellulitis" is rare, while abscess is relatively common. A further clinical observation is that the presence of *Cl. welchii* in a wound may delay healing; that is, a wound with a mixed infection of

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TABLE I.—Recorded Cases of *Cl. welchii* in Wounds
University of Alberta Hospital, 1955 - 1959

Simple contamination	40
Cl. welchii wound infection (Welch abscess)	16
Delayed healing (*Staph.* accompanying)	5
True gas gangrene	2
Total	63

Cl. welchii and *Staphylococcus aureus* may take longer to heal than one infected by *Staphylococcus aureus* alone.

In the University of Alberta Hospital over the five year period 1955-59, the presence in wounds of *Cl. welchii,* which is reported only if it occurs in apparently significant numbers or circumstances, was recorded in 63 cases. Its actual clinical significance is shown in Table I.

Most of these *Cl. welchii* infections followed abdominal operations. A detailed list of all *Cl. welchii* infections which followed various operations and traumatic wounds is shown in Table II.

TABLE II.—Detailed List of *Cl. welchii* in Wounds
University of Alberta Hospital, 1955 - 1959

Following ruptured appendicitis or acute suppurative appendicitis	
Number of infected wounds contaminated with *Cl. welchii*	6
Cl. welchii abscess	1
Following cholecystectomy or other operations on biliary tract	
Number of infected wounds contaminated with *Cl. welchii*	6
Cl. welchii abscess	2
Following gastrectomy	
Number of infected wounds contaminated with *Cl. welchii*	5
Cl. welchii abscess	2
Following operations on the ileum, colon or rectum (including Miles' resection)	
Number of infected wounds contamined with *Cl. welchii*	7
Cl. welchii abscess	1
Following herniotomy or orchidectomy	
Number of infected wounds contaminated with *Cl. welchii*	1
Cl. welchii abscess	2
Following suprapubic prostatectomy	
Number of infected wounds contaminated with *Cl. welchii*	2
Following burns	
Number contaminated with *Cl. welchii*	4
Following amputation for peripheral arterial disease	
Number of infected wounds contaminated with *Cl. welchii*	3
Cl. welchii abscess	1
Following traumatic injuries	
Number of infected wounds contaminated with *Cl. welchii*	6
Number of wounds in which *Cl. welchii* delayed healing	5
Cl. welchii abscess	5
True gas gangrene	2
Following miscellaneous conditions (hip pinning, pyelonephritis)	
Cl. welchii abscess	2

It is noteworthy that in all the infected wounds following gastrectomy the operation was carried out for *carcinoma.* There is probably more dead tissue in carcinoma suitable for the growth of anaerobic organisms, than there is in peptic ulcer. This is in keeping with the well known fact that wound infection is much commoner following gastrectomy for carcinoma than for peptic ulcer.

In nearly all of the above infections there were mixtures of organisms present including *staphylococci, E. coli, Aerobacter aerogenes, proteus, pseudomonas, paracolon, bacteroides* and *streptococci* or in other words intestinal organisms and pyogenic cocci, as well as *Cl. welchii.* However, in the cases of *Cl. welchii* abscess, this anaerobe was the predominating organism, and was thought clinically to be mainly responsible for the condition. As noted, it is most common following abdominal operations. One reason for this may be that though the antibiotics (such as neomycin) and sulfa drugs, commonly used to "sterilize" the bowel, have a wide antibacterial activity, they are only partially effective against *Cl. welchii,* a normal inhabitant of the bowel,[3] and certainly ineffective against *Cl. welchii* spores.

Clostridium Welchii Wound Infection (Welch Abscess)

The author has become increasingly familiar with this entity, if such it may be called, over the past five years. Sixteen cases were encountered, most of which developed following abdominal operations.

Clinically, the patient may have more fever than would be expected in the early postoperative period, and the pulse rate may be increased to 100 per minute. Signs of wound infection may be lacking in the first few days, but generally by the fourth or fifth day the incision shows a distinct bulge, with increasing redness and heat. The patient complains of pain in the wound. If a stitch is removed and the skin margins separated over the most bulging portion, much *foul-smelling, brownish*

watery pus exudes under considerable pressure, with perhaps a bubble of gas. With large amounts of this material and gas, one naturally suspects a bowel fistula. However, once the abscess is opened, temperature and pulse fall promptly to normal, the patient improves, and wound healing proceeds normally, though slowly.

In the 16 cases encountered, the abscess was usually confined to the subcutaneous fat. Cultures from the foul discharge yielded in a few cases *Cl. welchii* only, but in most, *Cl. welchii* plus mixed intestinal organisms. Penicillin was the commonest antibiotic used parenterally, and antitoxin was rarely administered, *but none of these patients developed true gas gangrene.* The cases occurred at wide intervals and in none was there any good evidence of cross infection.

Case Reports

Case 1.—Mr. P.S., aged 55 years had a subtotal gastrectomy in February 1958 for a large fungating carcinoma. His early course was uneventful. On the fifth day, however, his temperature was 101° F., pulse 100 per minute and he complained of pain in the wound. On inspection, the incision line was bulging, and when a stitch was removed, about 25 c.c. of foul smelling, watery, brownish pus escaped with a bubble of gas. This yielded a pure culture of *Cl. welchii.* Following drainage of the subcutaneous abscess, his temperature and pulse fell to normal the next day. The wound drained for about 10 days but was completely healed three weeks after the operation. This patient was given both penicillin and Chloromycetin®.

Case 2.—Mrs. I.K., aged 67 years, suffered a subcapital fracture of the right hip and a prosthesis was inserted on December 6, 1956. On the first postoperative day her temperature was 101° F., on the second postoperative day it was 102° F.

Four days postoperatively she showed a swinging temperature but otherwise she did not look particularly ill. Fluid and gas were palpated deep to the incision. Under general anæsthesia, a large foul-smelling abscess deep to the fascia lata was found. The muscle itself was alive and looked healthy. Direct smear showed Gram-positive bacilli and culture produced a pure growth of *Cl. welchii.* Penicillin was given. After drainage of the abscess her temperature subsided but she continued to run a low fever up to 100° F. for two weeks. The wound stopped draining after about two weeks.

Case 3.—Mr. P.D., aged 65 years had severe peripheral vascular disease, with early gangrene of a foot. Supracondylar amputation was performed on May 14, 1958, at which time the femoral artery was found to be occluded by thrombus but the muscle and skin bled.

On May 15 his temperature was 101° F. and there was some pain at the amputation site.

On May 16 his temperature was 102° F., pulse rate 120 and pain had increased. The dressing was taken down and the wound inspected. There was perhaps more swelling than normal but little drainage. A swab was taken however, for direct smear and culture. On May 17 his temperature was still elevated. A bacteriological report described Gram-positive bacilli on direct smear, morphologically resembling *Cl. welchii.* On May 18 his temperature was 102° F., pulse rate 120 and some crepitation was felt under flaps. This was confirmed by radiographs which showed gas located at the amputation stump site but not tracking along muscle planes (Figs. 1 and 2). It was thought that he had a *Cl. welchii* abscess localized to the stump. Cultures confirmed the presence of *Cl. welchii.* On May 19, the sutures were removed from the skin and fascia and the wound was left open. A moderate amount of foul-smelling, brownish, liquid material was present in the wound, but the underlying muscle appeared viable and retracted when cut. Repeat cultures on May 19 and May 21 also showed *Cl. welchii.* In 48 hours the patient's temperature and pulse returned to normal, and the wound infection at the stump gradually resolved by June 4, three weeks after his amputation. Since then, revisions of his amputation have been necessary in October 1958 and March 1959.

Delayed Healing Associated with Clostridium Welchii

Over the past five years, five instances have been encountered of delayed healing of traumatic injuries contaminated or "infected" with *Cl. welchii* and other organisms, usually *staphylococcus aureus.* Clinically these cases did not show *Cl. welchii* infection nor gas gangrene, and the diagnosis was made by finding the organism in considerable numbers on direct smear and in culture.

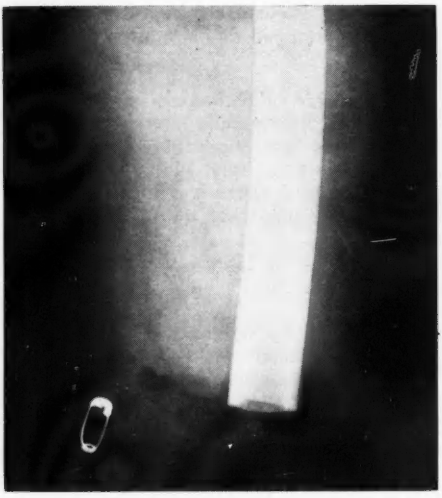


Fig. 1.—*Clostridium welchii* abscess localized to A.K. amputation stump four days postoperatively. Note the localized area of gas on radiograph.



Fig. 2.—Normal A.K. amputation stump four days postoperatively, for comparison. No gas is seen on radiograph.

Case 4.—Mr. W.T., aged 33 years, suffered a crushing injury to a thumb on September 24, 1957, with a fracture of the distal phalanx and some loss of skin. On October 1, a culture yielded *Staph. aureus* and *Cl. welchii*. By October 4, however, the wound looked clean enough for a partial debridement and secondary closure which was carried out. On October 6, the patient was acutely ill with a temperature of 103° F., and pulse rate 110, so the wound was opened and drained. He ran a fever of 101° F. to 102° F. for one week. Further operations for removal of sequestra and for debridement were required on October 27 and 29, and November 5.

He was finally discharged on November 11, seven weeks after his original injury. This case suggests the possibility that a wound cannot be closed with impunity if *Cl. welchii* is present.

Gas Gangrene

There is no infection from which man suffers, more terrifying than this. It is fearsome to patient and to doctor alike. Fortunately it is rare in civilian life. In this hospital we have had but five cases in the past 10 years, and only two in the past five years. Of the five cases, three followed traumatic injuries; two of these patients lived and one died. The fourth case followed infection and ulceration of thrombosed hæmorrhoids, and the last arose in the donor area, the ilium, for a bone graft of spine. Both of these patients died. The cases were all sporadic in nature, were not related in any way, and therefore no cross infection could be alleged.

External trauma, intrauterine trauma and strangulated hernia have accounted for most reported cases of gas gangrene.[4]

Clinically the disease pursues a rapid and fulminating course. Commonly appearing about the fourth, fifth or sixth day after injury, it may appear as early as seven hours or as late as six weeks. MacLennan[1] states that 65% of his cases developed gas gangrene within three days following war wounds. Death often supervenes a day or two after the diagnosis is made. Trauma that produces a bursting or shattering effect to tissues is particularly favourable to the infection, and the calf thigh and buttock are common locations.

The earliest symptom is pain at the site of the injury. This may be so much out of proportion to the early findings that the patient may be accused of malingering. On inspection, the wound may show only a little increased œdema with perhaps a tiny watery discharge. But within a few hours œdema progresses rapidly, and the

watery discharge becomes brownish. The skin surrounding the wound may show blebs filled with bloody fluid. If cultures are taken at this time, they must be obtained deep to any crusts or necrotic tissue. Immediate smear may show large Gram-positive rods, and anaerobic culture must be requested. Often there is very little or no gas, though usually a bubble of gas may be expressed from the wound. Serial radiographs, once or twice daily, may be helpful in demonstrating the presence of gas. Surgeons with experience claim they can "smell out" the process by the peculiar sweetish odour which is said to be characteristic.

Constitutional symptoms are those of a profound toxæmia. The temperature is often raised only slightly, but the pulse rate is usually rapid, and the blood pressure may fall, indicating severe shock. The patient may be pale and sweating, but still alert and bright eyed. Neither patient nor doctor may recognize the seriousness of the condition until the patient is *in extremis*, and death usually supervenes rapidly, despite treatment.



Fig. 3.—Gas gangrene left elbow region. Note the dirty wound with some suture material still present, the blebs filled with dark material, and the gross œdema of the skin.

The following case history is characteristic.

CASE 5.—Mr. A.L., aged 35 years, was involved in a car accident on May 4, 1957, and suffered a laceration of the left elbow penetrating into the olecranon bursa. The wound was debrided, sutured primarily, and he was given penicillin and antitetanus serum.

On May 6, there was some swelling of the elbow and he was given Chloromycetin® and Cathomycin®. On May 11 the whole arm was grossly swollen and the stitches were removed. The following day the patient was admitted to the University of Alberta Hospital, gravely ill with a temperature of 99° F., pulse rate of 100. He was frightened and toxic. The whole left arm was huge, from fingertips to shoulder girdle, at least twice normal size, and subcutaneous œdema had stretched the skin so that it resembled a plaster cast encircling the arm, threatening to cut off the circulation (Fig. 3). Through the œdema at the wrist, a good radial pulse could be felt. Over the olecranon, a thin brownish discharge exuded from beneath black necrotic skin. Anaerobic infection was considered likely. The process had extended to involve the whole left side of the thorax with œdema, so amputation was considered out of the question. Treatment was started with penicillin 500,000 units every three hours.

On May 14, 36 hours after admission, the whole arm was opened widely from shoulder to wrist, down to muscle level. The muscle did not appear necrotic. The olecranon bursa was opened widely. Cultures that day were reported as showing *Cl. welchii*. Despite blood transfusion, antitoxin and penicillin, the patient died rather suddenly on May 15, eleven days following his original injury. Autopsy revealed *Cl. welchi* present in muscle, extensive œdema of the whole left thorax as well as left arm, and bilateral pleural effusions.

The next case history is that of a man who survived.

CASE 6.—Mr. G.K., aged 24 years was run over by a truck on June 5, 1956. There was a crushing injury to the right thigh but no fracture. The femoral vein was torn but the artery was intact. A large skin flap, from knee to groin was sutured. The patient was admitted to hospital on June 7. Gas was found in the right thigh and could be traced subcutaneously to the right axilla. The whole right leg was discoloured. His temperature was 104° F., and pulse rate 130 per minute. Operation was carried out on June 8. The

sutures were removed revealing an evil-smelling purulent exudate. The adductor thigh muscles, which were necrotic, were excised. The right flank was incised and all wounds were packed widely open. Penicillin, 1,000,000 units, was given intramuscularly every three hours, and Terramycin® 500 mg. was given intravenously every eight hours. Direct smear of the pus showed large Gram-positive bacilli, which on culture proved to be *Cl. welchii*. On June 16, hæmorrhage from a branch of the right femoral artery was controlled by ligature. On June 24, a low thigh amputation was carried out, but on June 25 there was a hæmorrhage from the femoral artery, and to control it the external iliac artery was tied. The patient gradually improved, had a revision of his amputation stump on August 2, and was discharged finally from hospital, well, on October 4.

Case 7.—Mrs. L.P., aged 54 years, had a hysterectomy in 1956.* She was well until May 31, 1958, when she developed colicky abdominal pain, obstipation and vomiting which after a day or two became fæculent. On June 6 she was admitted to the Royal Alexandra Hospital, Edmonton, in fairly good condition considering that she had had almost complete intestinal obstruction for a week. The abdomen was moderately distended but showed no guarding and no rebound tenderness. Tinkling, high-pitched intestinal sounds were heard. Her temperature and pulse were normal and white blood count was 7000 per c.mm. A flat plate of the abdomen showed dilated loops of small bowel. Following supportive intravenous therapy and nasogastric suction for 12 hours, laparotomy was performed on June 7. Small bowel obstruction was found, caused by a small loop of ileum herniating through the broad ligament. This loop (4 cm. to 5 cm. in length) was fairly easily reduced, and was purplish in colour. Its normal colour returned rapidly and peristaltic waves passed through it, though it was noted that one of the mesenteric veins was thrombosed. It was thought wise not to do a resection and the abdomen was closed.

The patient was well for 18 hours following operation, after which time she became restless and hypotensive, with peripheral cyanosis. She was given neosynephrine, 1500 c.c. blood and Chloromycetin®, in the belief that her shock was most likely due to coliform septicæmia. Some gas developed in the laparotomy incision, and a little discharge appeared from which direct smear showed Gram-positive bacilli, which later proved on culture to be *Cl. welchii*. Her temperature was 100° F. and her pulse rate was 120 per minute. She died in a shock-like state on June 8, 25 hours after operation.

Autopsy showed peritonitis, and gas was demonstrated in the abdominal wall, liver and spleen. Cultures confirmed the presence of *Cl. welchii* in these sites as well as in the blood stream.

Looking back on this case, one wonders if the longstanding intestinal obstruction of a week's duration allowed virulent *Clostridia* to develop in the strangulated loop, which, when released (and following improvement of the blood supply), infected the peritoneal cavity and blood stream. Perhaps if the loop had been resected, this sequence might not have occurred. It was interesting that, at autopsy, the loop was still viable.

Treatment of Gas Gangrene

The prognosis is poor whatever treatment is used, and the mortality rate 50% to 75%. By far the most important prophylactic treatment is adequate wound debridement. Prophylactically, gas gangrene antitoxin is probably of little or no value, but penicillin may be.

Once the infection supervenes, measures must be heroic. Early amputation, if the process is confined to an extremity, or if not, wide drainage and excision of all dead muscle, are of the utmost importance. These wounds may be packed open with dressings saturated with zinc peroxide ointment, and crystalline penicillin G should be given in massive doses, 1,000,000 units intravenously and 1,000,000 units intramuscularly every three hours. Sensitivity tests in our laboratory, and also in reports from Korea,[5] suggest that the tetracyclines are the antibiotics of choice, so they should be given in full doses along with penicillin. They are probably best given intravenously, especially if the patient is in shock.

Theoretically, and also experimentally, gas gangrene antitoxin is useful in treating an established case.[6] Polyvalent (*welchii, septicum, novyi*) antitoxin is given both intravenously and intramuscularly in doses of 25,000 units every four hours.

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*I am indebted to Dr. H. Richard, Royal Alexandra Hospital, Edmonton, for this case report which shows how gas gangrene may very rarely (fortunately) result from strangulation of the bowel.

For a patient in shock, as most of these cases are, intravenous hydrocortisone, in the form of Solu-Cortef®, 100 mg. every six to eight hours may be of some use, to alleviate depleted adrenocortical function.

General supportive measures should be pursued actively. These include *multiple* and sometimes *massive* blood transfusions, sedation for pain, and care of fluid and electrolyte balance, watching especially for acidosis.

Discussion

From the review of all *Cl. welchii* infections in a large hospital over the past five years, certain conclusions may be drawn. Simple wound contamination is relatively frequent and was noted in 40 cases, even though it was reported by the laboratory only when it was considered likely to be of significance. Trivial contamination is undoubtedly much more frequent. In five cases, the presence of *Clostridia* probably delayed wound healing. Rather surprisingly, an entity of *Cl. welchii* wound infection (or Welch abscess) is not rare, since 16 cases were encountered. In these patients, there is no grave problem, for simple drainage cures the infection in most instances quite speedily. There is no need for undue concern, clinical or administrative, about this type of case, for it appears to have no relationship to gas gangrene.

In this series not a single instance of cross infection has been found. These cases are no more infectious than a coliform infection, for example, and decidedly less so than staphylococcal (especially due to so-called hospital strains) or hæmolytic streptococcal infections.

True gas gangrene in hospital practice appears to be a sporadic, rare, but catastrophic event. Only two cases were seen in a five year period. We doubt if this incidence can be lowered by strict isolation of all cases of *Cl. welchii* contamination and infection, and indeed our experience denies this, for none of these cases went on to develop gas gangrene, and none "spread" gas gangrene to a new patient. Since some of the cases of "Welch abscess" were nursed with isolation technique, this conclusion might not be altogether valid. However, in both of the patients who developed gas gangrene, there was inadequate wound debridement and the wounds were closed primarily. Both in reality were admitted with gas gangrene.

The only really effective prophylactic measure against the development of gas gangrene is thorough cleansing and debridement of "dirty" wounds, leaving them open. This has been proven conclusively in both World Wars, and in Korea, but is sometimes forgotten in civilian life. It is realized that many wounds are better closed primarily, such as those in the face, and over tendons.

Why an occasional wound, adequately treated, will develop gas gangrene, and 50 similar ones will not, is still an unanswered problem. It must have something to do with the virulence of *Cl. welchii*, and the resistance of the patient.

Summary

An attempt has been made to classify and investigate all cases of *Cl. welchii* infection recorded in a hospital over the past five years. Simple contamination is relatively common, "Welch abscess" not uncommon, and true gas gangrene decidedly rare. As far as we can ascertain, contamination and wound infection rarely if ever, lead to gas gangrene. We feel that none of these infections are very "infectious" as far as other hospital patients are concerned, because we have not noted cross infection, when no special precautions were taken.

The finding of *Cl. welchii* in a wound has no particular significance and demands no greater administration or nursing attention than that accorded to any septic wound. "Witch hunts" are off! Gas gangrene is a clinical and not a bacteriological entity.

The treatment of *Cl. welchii* abscess, or wound infection, is simple drainage; the treatment of gas gangrene is heroic, with radical surgery, antibiotics, gas gangrene antitoxin, blood transfusions and other supportive measures. Since the mortality rate or gas gangrene remains so high, prophylaxis is all important, and consists of adequate cleansing and excision (debridement) of wounds.

Acknowledgment

Dr. R. D. Stuart, Professor of Bacteriology, University of Alberta, has accompanied the writer

on "infection rounds" of the surgical and pædiatric services of the University of Alberta Hospital, once weekly over the past five years. The hospital is grateful for his keen interest in clinical bacteriology, and I am deeply indebted to him for his stimulating criticism of this article.

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Résumé

Une tentative pour classifier et investiguer tous les cas d'infection par *Cl. Welchii* survenus à l'Hôpital de l'Université d'Alberta durant les derniers cinq ans, a été entreprise. La simple contamination est assez fréquente, l'abcès à *Cl. Welchii*, non inusité, tandis que la gangrène gazeuse est décidément rare. En autant qu'on puisse le certifier, la contamination et l'infection des plaies conduisent rarement, à la gangrène gazeuse. Nous avons l'impression qu'aucune de ces infections n'est contagieuse, en ce qui concerne les autres malades hospitalisés, n'ayant jamais relevé d'infections transmises, alors qu'aucune précaution spéciale n'était encouragée.

L'identification de *Cl. Welchii* dans une plaie n'a pas de signification particulière, ni demande de plus grande précaution d'administration ou de "nursing" que celle accordée à toute plaie infectée. La gangrène est une entité clinique plutôt que bactériologique.

L'abcès *Cl. welchii*, se traite par simple drainage; la gangrène gazeuse se traite héroïquement par une chirurgie radicale, par l'administration d'antibiotes et d'antitoxine et par des transfusions sanguines associées aux autres médications de support.

Puisque le taux de mortalité dans la gangrène gazeuse reste élevé, la prophylaxie est des plus importante, et consiste en un nettoyage adéquat et en un débridement de plaies.

THE GENERAL PRACTITIONER

ROGER GILES, SURGIN PARISH CLARK AND SCHULEMASTER, GROSER AND HUNDERTAKER*

Respectfully informs ladys and gentlemans that he drors teef without waiting a minute, applies laches every hour, blisters on the lowest tarms, and vissicks for a penny a peace. He sells Godfathers kordales, kuts corns, bunyons, docters hosses, clips donkies wance a month, and undertakes to took after everybody's navls by the ear. Joesharps, penny wissels, brass kanelsticks, frying pans, and other moosical hinstruments hat greatly reduced figers. Young ladies and gentlemen larnes their grammur, and langeudge in the purtiest manner, also grate care taken off their morrels and spelling. Also zarm singing, taychıng base vile and other sorts of fancy work, squadrils, pokers, weazels, and all country dances tort at home and abroad, at perfeksun. Perfumery and snuf in all it branches. As times is cruel bad I beg to tell ee that i has just beginned to sell all sorts of stashonery, ware, cox, hens, vouls, pigs and all other kinds of poultry, blackin-brishes, herrins, coles, scrubbing-brishes, traykel and godly bukes and bibles, mise-traps, brick-dist, whisler-seeds, morrell pokkerankechers, and all sorts of swatemaits, including taters, sasages, and other garden stuff, bakky, zizars, lamp oyle, tay kittles and other intoxzikating likkers, a dale of fruit, hats, zongs, hair oyle, pattins, bukkets, grandin stones and other aitables, korne and bunyon zalve, and all hardware. I has laid in a large assortment of tripe, dogs mate, lolipops, ginger beer, matches, and other pikkles, such as hepson salts, hoysters, Winser sope, anzetrar—Old rags bort and sold here and nowhere else, new laid eggs by me Roger Giles; zinging burdes keeped, such as howles, donkeys, paykox, lobsters, crickets, also a stock of celebrated brayder.

P.S.—I tayches geography, ritmitmetic, cowsticks, jimnastics and other cheynees tricks.

*Exact words of an old Cornish signboard.

URINARY DIVERSION TO THE ISOLATED ILEAL SEGMENT*

A. D. MacKENZIE, M.D., F.R.C.S.[C] and
G. J. ANKENMAN, M.D., F.R.C.S.[C],† *Vancouver, B.C.*

URETEROINTESTINAL anastomosis has been carried out by various techniques since the latter 19th century, in an attempt to perfect a satisfactory substitute for the urinary bladder. Clarke and Leadbetter have surveyed the results of 2897 ureterosigmoilostomies alone.[1] This procedure carries a high incidence of reflux with hyperchloræmic acidosis in a third of cases irrespective of technique, and ascending pyelonephritis in over 30%. Although these hazards are reduced using a combined tunnel principle and end to side anastomosis, it is now generally felt that the urinary and fæcal stream should usually be separated to achieve long term satisfactory results. Two methods are in common usage at the present time; the rectal bladder (dry colostomy with ureterosigmoidostomy), and urinary diversion to an isolated segment of ileum.

The rectal bladder provides a low pressure and usually sterile reservoir for urine. Differential electrolyte absorption may occur, but is not often a clinical problem in absence of renal damage. Reflux does occur, but under a considerably lower pressure than when bowel continuity is intact.[2] A competent rectal sphincter is essential for continence. The majority apparently do have some difficulty with enuresis.[3]

The use of an isolated segment of ileum is an attractive alternative for urinary diversion. Fifty years ago Shoemaker successfully treated a patient with a solitary kidney and tuberculous bladder by diverting the ureter to an ileal loop brought to the skin surface. Later the ileum was rediverted into the bladder. Since 1950 Bricker has been a leading proponent of this method of urinary diversion on this continent and has reported on a large series of such cases at various times.[4] Excellent contributions on the advantages and disadvantages of the use of ileum in urological surgery have been made by Pyrah.[5, 6] The essential advantage of ileum is that active peristalsis tends to keep the segment empty[7] when brought to the skin surface so that it acts as a conduit rather than a reservoir reducing stasis and subsequent ascending infection and electrolyte reabsorption. The main disadvantage is the necessity of wearing a permanent urinary drainage appliance.

The first ureteroileal conduit operation was performed in Vancouver in 1951 on a young man who was incontinent of urine from congenital disease. At the present time he is gainfully employed. Blood chemistry is within normal limits, and the upper urinary tract is normal on intravenous pyelography. It is our purpose in this paper to review the indications for the procedure carried out in 52 cases at Shaughnessy Military Hospital and the Vancouver General Hospital since 1951, and to discuss the technique and results in 35 cases in which first-hand knowledge and complete follow-up is available.

The indications for this procedure are listed in Table I. There are many possible indications. The procedure is particularly applicable to congenital lesions associated with an incompetent anal sphincter where the necessity for urinary diversion exists.[8]

Good illustrated papers describing the technical aspects of the procedure are available in the literature.[4-6] With experience, some useful modifications have been made.

The gastrointestinal tract is mechanically prepared in the usual manner. Although bowel sterilization was carried out in earlier cases, it is no longer considered necessary or advisable.

TECHNIQUE

The abdominal incision is planned in accordance with the exposure needed, leaving the right abdomen unscarred for application of appliance. A lengthy left lower paramedian incision will provide

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*Presented by Dr. G. J. Ankenman at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, January 1960.

†University of British Columbia and the Vancouver General Hospital.

TABLE I.—Indications for Ureteroileal Conduit Operation

	No. of patients
I. *Congenital*	
1. Exstrophy of bladder	2
2. Recto-vesical fistula	2
3. Myelo-meningocele	1
II. *Inflammatory*	
1. Contracted bladder and hydronephrosis of tuberculous origin	2
2. Intractable urethral stricture	1
III. *Neoplastic*	
1. Carcinoma of urinary bladder	
(a) palliative diversion	8
(b) preparatory to cystectomy: completed	7
contemplated	2
(c) coincidental with cystectomy	9
2. Carcinoma of urinary bladder and prostate	1
3. Carcinoma of cervix.	
(a) fistula (post-radiation)	6
(b) intractable radiation cystitis and recurrent pyelonephritis	1
(c) coincidental to pelvic evisceration	1
4. Carcinoma of rectum invading G.U. tract.	4
5. Carcinoma of female urethra	1
IV. *Neurogenic bladder*	4
Total	52
Included in the above series:	
1. Conversion of ureterosigmoidostomies	
(a) recurrent pyelonephritis	1
(b) recurrent pyelonephritis and hyperchloræmic acidosis	2
(c) progressive hydronephrosis	1
2. Conversion of nephrostomy solitary kidney (local pain)	1
3. Conversion of ileocystostomy (Cordonnier procedure)—surgical failure	1

good exposure. An appendectomy is carried out at a suitable stage to avoid future diagnostic and technical problems associated with appendicitis. The ureters are isolated early below the pelvic brim and temporarily ligated to distend them with urine. When a cystectomy is performed it is probably desirable to remove the bladder first to avoid traction on anastomotic sites.

A segment of ileum, 12 cm. to 20 cm. in length is selected 30 cm. to 45 cm. proximal to the ileocæcal valve. The loop should not

TABLE II.—Sex and Age Distribution—Both Sexes, (Men 36—Women 16)

Age	*No. of paitents*
0 - 10	4
20 - 30	2
30 - 40	4
40 - 50	6
50 - 60	12
60 - 70	17
70+	7
Total	52



Fig. 1.—Isolation of the ileal segment.

be made longer than necessary to pass through the abdominal wall with a broad mesenteric base (5 cm. to 7 cm.) which is not incised deeply (4 cm. to 5 cm.) shown in Fig. 1. Transillumination will aid in choosing a segment with a good arterial arcade. Bowel continuity is reestablished anterior to the loop by end to end anastomosis (Fig. 2). The mesenteric rent is closed with fine silk sutures with care not to close the apex too tightly and compromise the blood supply to the isolated segment. The loop is irrigated and the proximal end closed (Fig. 3).

The ureters which are now distended with urine are mobilized with care to pre-

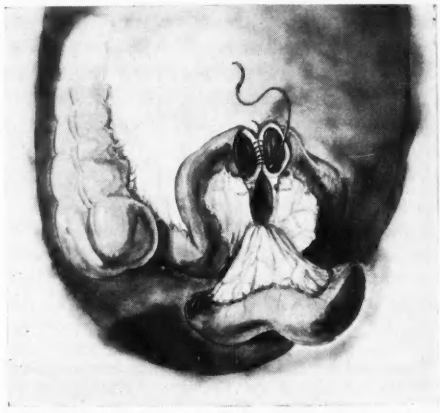


Fig. 2.—End to end anastomosis anterior to the isolated segment.

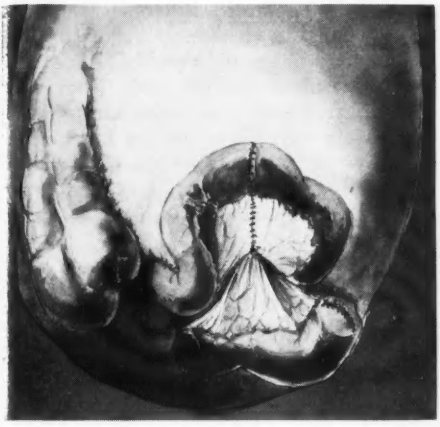


Fig. 3.—Closure of proximal end of segment.

serve the adventitia. The left ureter is brought through the sigmoid mesentery without angulation (Fig. 4). The ureteral ends are "fish-mouthed" and anastomosis is carried out end to side on the antimesenteric border adjacent to the proximal end of the loop. Anchor sutures are placed at each corner and mucosa to mucosa anastomosis is completed using continuous 0000 chromic catgut (Nesbit technique).



Fig. 4.—Left ureter brought through sigmoid mesentery without angulation.

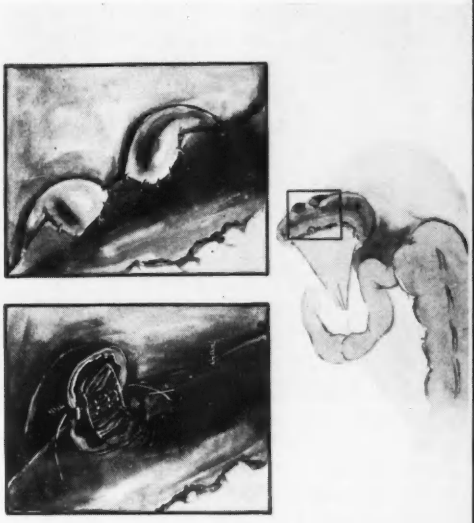


Fig. 5.—Ureteroidal anastomosis.

A minimal number of interrupted sutures may be used for reinforcement if necessary (Fig. 5). The anastomosis is facilitated if done over a red rubber catheter which is withdrawn at completion.

A suitable ileostomy site is selected between the right anterior superior iliac spine and umbilicus. There must be adequate room for the disc of the permanent urine receptacle. Leakage can be a problem if the disc overlies an operation scar or a low costal margin. A circular piece of skin 1 inch in diameter is excised along with the underlying subcutaneous tissue and external oblique fascia. An adequate aperture is made through the remaining abdominal wall and peritoneum and the distal end of the ileal segment is brought to the skin surface. An ileal bud about 1 cm. in elevation is created using four silk everting sutures incorporating the (a) bowel mucosa (b) seromuscular coat 2.0 cm. proximal (c) margin of skin aperture. The spaces between these major sutures are closed by approximating mucosa and skin (Fig. 6). Finally, the ileal loop and ureters are tacked to the peritoneum to close all apertures. A drain brought out through a stab wound is essential as urinary drainage is not uncommon for a few days. No. 30



Fig. 6.—Formation of ileostomy bud.

wire is used for fascia in abdominal closure. The skin about the ileal bud is prepared with tincture of benzoin compound in the operating room, and a Thomas Fazio Limited drainage apparatus is applied snugly about the bud. This permits inspection of the bud, is atraumatic and serves quite satisfactorily until the permanent appliance is fitted in two to three weeks' time. Catheterization of the loop has not been necessary.

Postoperative Care

Continuous gastric suction is necessary for a longer period than usual after abdominal surgery. It is ordinarily four or five days before active peristalsis is present. This may, in part, be due to ileal anastomosis and partly due to urinary leakage. Electrolyte replacement and antibiotic administration are necessary.

Ileostomy bags should be changed as soon as they begin to leak, in order to protect the skin. In the first half of the series, ileostomy dilatations were carried out using the finger, but recently this has not been done and retraction stricture has not been a problem. Before discharge from hospital the patient is fitted for a permanent urine receptacle* as designed by Nicolai.[9] A set of discs with various sized apertures and concavities is available for individual fitting and the appliance ordered accordingly. This apparatus can be worn up to 10 days without change.

Adequate follow-up should include periodic electrolyte studies and intravenous urography. There is always early hydronephrosis ostensibly related to anastomotic œdema, and little is to be gained by radiography until six weeks postoperatively unless there is a clinical indication.

Postoperative Results

Complications, present status and classification of results are tabulated in Tables III, IV and V. In 50% of cases no complications were recorded.

TABLE III.—Complications in 35 Patients on Whom Ureteroileal Conduit Operation was Performed

General	
I. *Early*	
Coronary insufficiency	1
Thrombophlebitis	1
Atelectasis	1
Pneumonia (*staph. aureus*)	1
Unexplained jaundice	1
II. *Late*	
Homologous serum jaundice	1
Specific	
I. *Early*	
Intraperitoneal	
Prolonged paralytic ileus	2
Mechanical obstruction requiring surgical relief	2
Urinary fistula	3
Fæcal fistula (leading to peritonitis and death)	1
Wound	
Infection (all minor)	7
Dehiscence	1
Acute pyelonephritis	1
II. *Late*	
Electrolyte disturbances	6
Ileal bud	
Redundancy	1
Retraction and stricture	1
Ulceration	1
Difficulty fitting appliance	2
Hydronephrosis	5
Urinary calculi	
Renal and loop calculus	1
Ureteral	1
Wound	
Ventral hernia	1

TABLE IV.—Present Status

Surgical deaths	1
Dead of primary disease	8
Dead of uræmia	1
Alive and well (no known disease)	20
Alive (known disease)	5
Total	35

*Supplied by Eastern Sales Co., 1559 New Britain Avenue, West Hartford 10, Connecticut.

TABLE V.—Classification of Results

Good	28
Poor	4
Equivocal	3
Total	35

Morbidity and Mortality

There was one operative death in the series of 35 patients (mortality rate 2.8%) due to a technical error when, during closure a loop of bowel was caught by a suture with subsequent small bowel fistula. This occurred in an elderly man with poor resistance and he died of generalized peritonitis. This mortality rate is lower than that of most reported series. (Pyrah—25% mortality in 212 cases, Cordonnier[10]—9% mortality in 41 cases, Bricker[4]—over 10% mortality in 150 cases, but many of these deaths were attributable to pelvic evisceration and when figures are broken down it is considered that the mortality rate from ileal segment procedure should be about 3%).

General complications were not greater than might be expected with any major abdominal surgery. Two cases of mechanical small bowel obstruction required surgical relief. In one case obstruction was at Lane's band, the other was due to a twisted loop. Although some urinary drainage for a short time is not uncommon, in three instances it was prolonged for a few days with spontaneous resolution.

In this centre even a stitch abscess is classified as a wound infection. In no instance was infection serious or was hospitalization prolonged as a result. There was one instance of wound dehiscence although in other series this is not an uncommon complication (Pyrah 14%, Cordonnier 9%). Our low incidence may be related to almost routine use of wire for fascial closure.

Late electrolyte abnormalities were notable in six cases. Two of these patients (converted ureterosigmoidostomies) had renal damage preoperatively and required medication. In general, conversion of ureterosigmoidostomies to an ileal bladder improved the electrolyte disturbance. Two other patients who eventually died of their primary disease had disordered electrolytes. Two are alive and well with a mild asymptomatic hyperchloræmic acidosis and are not on medication.

A redundant ileostomy which was made too long initially was satisfactorily modified under local anæsthesia. A retracted and relatively strictured ileostomy in one patient does not require correction. Two patients have had difficulty with appliance fitting and subsequent leakage. In one instance this is due to the appliance disc overlying a low costal margin and in the other the appliance overlaps an old appendectomy scar.

Out of eight cases of well developed preoperative hydronephrosis there was complete regression by diversion of both ureters in three patients. In two of these there was no demonstrable function on intravenous pyelography preoperatively. For that reason we have no hesitation in anastomosing markedly dilated ureters unless a ureter is grossly so unhealthy that anastomosis is likely to fail. Such a ureter was tied off uneventfully on four occasions. In one patient a non progressive hydronephrosis has remained. In five cases postoperative hydronephrosis developed. Four of these patients died of their primary disease. The other patient developed a stricture at the anastomotic site and required an emergency nephrostomy. Re-anastomosis was later carried out with a satisfactory result.

The preoperative pyelogram shown in Fig. 7a is that of a patient who had a ureterosigmoidostomy five years previously because of radiation fistula. She had recurrent pyelonephritis for three years before conversion to ileal loop drainage. The upper tract showed good recovery within two months (Fig. 7b).

One patient with a solitary kidney has developed renal calculi, but renal reserve is so poor that surgery is not contemplated. A relatively large calculus demonstrated in his ileal loop by radiography was crushed under direct vision with a lithotrite. Another patient was admitted to hospital in shock secondary to septicæmia. The right ureter was found blocked by a calculus. Following ureterolithotomy he has done well.

It is not easy to categorize results, but from the point of view of patient's satisfaction and pyelography in 28 cases the



Fig. 7a.—Preoperative intravenous pyelogram.



Fig. 7b.—Two months postoperative intravenous pyelogram.

results are good or at least satisfactory. In four cases in which results are categorized as poor, all died of their primary disease and although the diversion possibly prolonged life for a short time, their disease was too advanced to have made the procedure worthwhile.

Conclusions

A review of urinary diversion to the isolated ileal segment in 35 cases has been presented. There was one operative death due to a technical error. Overall morbidity approaches 50%, but with experience this has been reduced. The results indicate that this method of urinary diversion is a good one even for palliative purposes. It is a major surgical procedure requiring close attention to details for satisfactory results. It should not be contemplated on the debilitated patient with advanced disease and a short life expectancy. More time and experience are perhaps required to decide if it is a superior diversionary method to the rectal bladder. It appears to be the method of choice where an incompetent anal sphincter exists. In the presence of hydronephrosis it would seem more physiological to divert the urinary stream to a conduit rather than a reservoir behind a functioning sphincter. Finally, the ileal bladder allows the satisfaction of a normal bowel movement which can be one of the highlights of an older individual's existence.

Acknowledgment

The authors thank Mr. Victor Doray, Medical Illustration Department, Vancouver General Hospital, for his assistance in preparation of the illustrations.

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RÉSUMÉ

Plusieurs techniques d'anastomose urétéro-intestinale ont été proposées, depuis la fin du 19ème siècle, dans le but d'obtenir un substitut satisfaisant pour la vessie. Les deux méthodes qui dominent actuellement sont la vessie rectale (colostomie avec urétéro-sigmoïdostomie) et la vessie iléale.

La vessie rectale procure un réservoir habituellement stérile et à basse pression qui ne crée pas de problème électrolytique si les reins sont sains. La continence est assurée par la compétence du sphincter anal.

L'utilisation d'un segment d'iléon isolé constitue une alternative attrayante de dérivation urinaire. Depuis 1950, Bricker s'est fait le propagandiste de cette méthode et a rapporté plusieurs séries de cas. L'avantage essentiel de cette méthode vient de ce que, du fait du péristaltisme actif, le segment d'iléon tend à se vider et qu'étant abouché à la peau, il devient un conduit plutôt qu'un réservoir, réduisant ainsi la stase et l'infection ascendante subséquente, de même que la réabsorption électrolytique. Le principal désavantage vient de la nécessité de porter un appareil en permanence.

L'expérience de 52 cas au "Shaughnessy Military Hospital" et au "Vancouver General Hospital", depuis 1951, associée aux résultats de 35 malades, dont l'évolution post-opératoire est connue, montre que cette méthode est adéquate, même dans un but palliatif. L'intervention est grave et requiert une attention particulière des détails pour l'obtention de résultats satisfaisants.

Elle ne doit pas être entreprise chez des malades gravement atteints et dont les jours sont comptés. Le temps et l'expérience montreront si la vessie iléale est meilleure que la vessie rectale. Elle semble indiquée dans les cas d'incompétence anale. En présence d'hydronéphrose, il semblerait plus physiologique de dériver le flot urinaire à travers un conduit, que dans un réservoir fermé. De plus, la vessie iléale, permet l'évacuation intestinale normale.

ATLAS OF ANATOMY AND SURGICAL APPROACHES IN ORTHOPÆDIC SURGERY —UPPER EXTREMITY. Vol. I. Rodolfo Cosentino, M.D., Preface by Arthur Steindler. 192 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $11.50.

The author has prepared an atlas of the anatomy of the upper extremity that is of interest in orthopædic surgery. Using photographs of carefully dissected specimens, the various regions of the upper extremity are well visualized and labelled. Minor structures are not labelled, allowing for clarity of the figures. The immediate relationships of important structures are demonstrated in each dissection. Figure 29 is especially good, showing the ulnar nerve as it passes through the medial intermuscular septum.

The second part of each region deals with the surgical approaches to this area. This emphasizes the specific anatomy of the incision. However, the very nature of this volume allows for only a few incisions to be illustrated, leaving the "Atlas of Surgical Approaches" totally incomplete. This must not be allowed to detract from the value of the work. The utility of this book is its clarity of appreciation of structures that are important, and this constant clarity and appreciation is demonstrated throughout the entire volume.

The author is to be congratulated on his publication of a work requiring such meticulous attention to detail. Surgeons who wish to review the anatomy of a region, will find this volume easy to use and satisfying in content.

A DICTIONARY FOR MEDICAL SECRETARIES. Isabel Alice Stanton, Secretary, Director of Graduate Training, Baylor University College of Dentistry. 175 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.25.

As the author states "If I were beginning again as a secretary, the very first thing I would do would be to learn the word roots". In the reviewer's opinion, doing this and, of course, everyday practice in the use of medical terminology are the fundamentals most relied on by the medical secretary.

The larger well known medical dictionaries, although quite suitable for the medical secretary as far as spelling and pronunciation are concerned, can hardly be termed simple. The definition of words in "A Dictionary for Medical Secretaries" will certainly present a much clearer picture than in the past, for the experienced medical secretary, as well as a fairly simple basic dictionary for the novice. However, it must be noted at this point that the Canadian secretary uses the English style of writing certain words and not the American style.

Irregular Words (which often present problems) and the List of Instruments given, are again examples of how successfully the author has managed, in so compact a dictionary, to cover the most important information required while structuring it in such a way as to take in the medical terminology that the medical secretary will be called upon to use most frequently.

RECENT ADVANCES IN HYPOTHERMIA*†

W. G. BIGELOW, F.R.C.S.[C] and R. O. HEIMBECKER, F.R.C.S.[C],‡ *Toronto, Ont.*

THE PURPOSE of this paper is to review some refinements in the technique of pure hypothermia and outline our current application of hypothermia combined with cardiac bypass. One clinical example of the use of pure hypothermia and one example of cardiac bypass combined with hypothermia will be discussed.

SIMPLE HYPOTHERMIA

Clinical hypothermia in the range of 28° C. to 31° C. has become a safe procedure provided four essential problems are medically managed. These will be discussed briefly.

1. *Control of Body Temperature*

Surface cooling is preferred to extracorporeal cooling because it is easier. In children a cold water bath, or ice bags are used. In large children and adults, cooling blankets are still our method of choice. This should be carried out in an air conditioned operating room (less than 22° C.) with six ice bags inside the blanket. It is simpler and just as efficient as immersion in cold water. Besides experience and a knowledge of drift in body temperature, we are guided by the rectal and œsophageal temperature. Fig. 1 illustrates the relationship between these two temperature readings during an open heart operation. The œsophageal thermocouple should lie opposite the heart. An indication that the drift in body temperature after active cooling is near an end is the reduction in the temperature difference between œsophageal and rectal reading which occurs.

Cooling machines are available with a separate warming reservoir which allows rapid switchover to rewarming.

2. *Cardiac Irritability*

The use of prostigmine with circulatory arrest has been important in reducing cardiac irritability. As the temperature falls below 30° C. however, there is increased cardiac irritability. This was originally blamed on the low blood pH but the study by Waddell[1] in our laboratory demonstrated a surprising relationship between the blood citrate level and the incidence of irreversible ventricular fibrillation during surgery. This is shown in Table I and from this table it may be seen that the use of citrated blood produced a high incidence of irreversible fibrillation. When this is compared with operations carried out with heparinized blood one notes the expected low citrate levels and that irreversible ventricular fibrillation has not been a problem. We now use fresh heparinized blood and at proper body temperature levels the heart does not show undue irritability. During closure of the thoracotomy wound protamine sulphate is given in dosage equal to the amount of heparin administered.

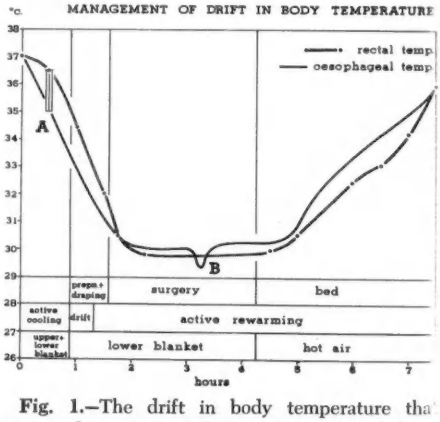


Fig. 1.—The drift in body temperature that occurs after active cooling is accompanied by a gradual reduction in the difference between rectal and œsophageal temperature. When these two are equal it usually indicates a stable body temperature.

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*Presented to the International Cardiovascular Society, Munich, Germany, September 1959.

†This work has been supported by grants from: The Ontario Heart Foundation, the Bickle Foundation and the Department of National Health.

‡Cardiovascular Division, Toronto General Hospital.

TABLE I.—Control of Cardiac Irritability Hypothermia—42 Cases

	Serum citrate over 20 mgm. %	*Death due to venticular fibrillation*
Citrated blood.....	40%	16%
Heparinized blood....	0%	0%

3. *Rewarming Shock*

The occasional appearance of shock and death during rewarming was a problem. In Waddell's study it was found that this was related to a metabolic acidosis which could not always be corrected by hyperventilation or respiratory alkalosis. This work showed that rewarming shock could be eliminated with better control of pH if one or two types of anæsthetic was used. These were (1) lytic cocktail or (2) the addition of ether during the period of cooling. Shivering was thus controlled. Table II illustrates these points. We prefer the ether technique and this has been described by Virtue.[2]

4. *Occasional Bleeding Tendency with Hypothermia*

Occasionally following hypothermia one encounters a patient, usually an adult, with a decidedly abnormal tendency to bleed. The one consistent abnormality of the blood which might explain this has been the development of thrombocytopenia during cooling.[3] Wensel[4] has studied this phenomenon and by administering 15 mg. to 20 mg. of heparin intravenously before human cooling we have reduced the thrombocytopenia during hypothermia, from 43% to 19%. Since using this technique in the last 30 open heart operations there has been no evidence of abnormal bleeding.

It is interesting that this bleeding tendency does not seem to be as great a problem in the hypothermia surgery of children. This may be explained by some essential difference in their platelets.

TABLE II.—Control of Rewarming Shock Hypothermia—29 Cases

Method of anæsthesia	*pH below 7.20*	*Rewarming shock*
Pentothal, nitrous oxide, curare.....	50%	40%
Lytic cocktail or ether...	21%	0%

Clinical Application

The Correction of Septum Secundum Atrial Septal Defects

Pure hypothermia still has a place in open heart surgery in view of recent improvements in diagnostic techniques. The claim that all congenital defects should be corrected with cardiac bypass so that the surgeon is "in a position to deal with unexpected pathology" is currently not tenable. Vector electrocardiography, better selective dye curves and angiocardiography have made it possible to diagnose uncomplicated atrial septal defects with great accuracy.

At the Toronto General Hospital, 50 adults with atrial septal defects of the secundum type have been operated upon using pure hypothermia. A survey of these patients, 15 years to 57 years of age, is shown in Table III. There has been only one death in the elective group of 42 cases, a mortality rate of 2.4%.

In a salvage group of eight cases with either total pulmonary resistance of 800 or over, or terminal cases in chronic, uncorrectable failure in hospital, there were four deaths. The average age in this group was 41 years, with three over 50 years of age.

Thus the correction of an uncomplicated atrial septal defect is safe and is indicated once the diagnosis has been made, provided there is at least a 2 to 1 pulmonary to systemic flow ratio.

Patients with atrial septal defects which we now prefer to repair with cardiac bypass are those with: (1) anomalous pulmonary venous drainage, particularly associated with the superior vena caval type defect; (2) associated mitral valve involvement; (3) where there is possibility of a primum or atrio-ventricular communis type of defect.

Twelve septum secundum defects have been operated upon with cardiac bypass.

TABLE III.—Atrial Septal Defect—Adults (Secundum)
50 Operations: Hypothermia

Cases	*Number*	*Deaths*	*Mortality percentage*
Elective.....	42	1	2.4
Terminal.....	8	4	50.0

Nine were found to have complicated defects at operation and there were no deaths.

All patients with atrial defects treated by both methods have been tested postoperatively with dye curves, or right heart catheterization. These results will be reported. As in other reported series,[5, 6] there has been an improvement in the mortality rate and there have been no deaths in this series in the last two years.

Combined Hypothermia and Cardiac Bypass

Clinical Application

Our clinical cardiac bypass surgery has been combined with hypothermia in 88 of the last 95 operations. A heat exchanger has been designed by Doctor Heimbecker,[13] a modification of the Duke University unit,[7] and it has been used on the arterial side of the circuit which includes a modified Cooley bubble oxygenator and a Pemco pump. A body temperature of 28° C. to 31° C. during cardiac bypass has increased the efficiency of flow rates. This technique is used on all types of cases unless a short bypass is expected. It has been accompanied by a lower incidence of pulmonary complications, more satisfactory blood pressure following bypass and a smoother postoperative course than previously observed. Swan[8] has reported that at a body temperature of 30° C. only about one-half the flow is required to maintain a constant arteriovenous oxygen difference. Anoxic arrest when required should be better tolerated.

The interior of the heat exchanger has many highly polished, thin walled, steel tubes. The temperature of the circulating water is simply controlled by a thermostat which in turn is connected to the hot and cold water tap. The complete unit is shown in Fig. 2. The cooling process takes about five minutes to attain body temperature of 29° C. to 31° C.[13] Thus it does not need to increase the complete bypass time. It is initiated during partial bypass and is completed while on total bypass. The inferior vena caval cannula may be partially occluded while on total bypass to ensure more general cooling. The desired temperature has usually been attained by the time the cardiac lesion has been examined by cardiotomy.

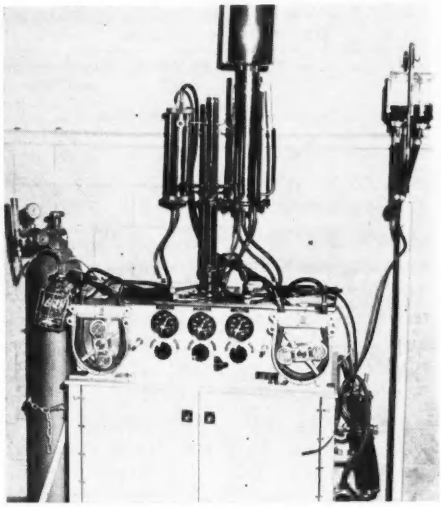


Fig. 2.—Combined hypothermia and cardiac bypass. A heat exchanger (left) is connected to modified Cooley oxygenator (right) and Pemco pumps mounted on wagon (lower portion of illustration).

More caution is necessary during rewarming and a maximum temperature in the heat exchanger of 40° C. produces slower rewarming. Since this takes a somewhat longer time, and to ensure that it does not significantly add to the complete bypass time, rewarming is instituted towards the end of the technical suturing process and when completion is assured.

The Surgical Correction of Mitral Regurgitation

Our small series of 16 cases of open heart annuloplasty for mitral regurgitation not only illustrates the application of this combined technique but the operation is of some current interest in itself. There are two essentials to the investigation of cases of mitral regurgitation. Left heart catheterization with two needle technique of reflux dye curve has been carried out in 50 cases in our cardiovascular unit[9] and has proven to be a very reliable guide to accurate diagnosis. A forward dye curve with oximeter in the ear is combined with a reflux curve from a cuvette recording of left atrial blood. Analysis of these curves

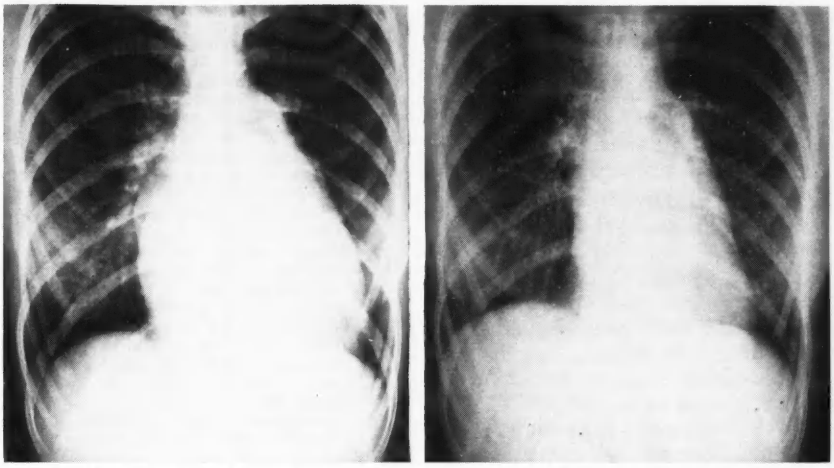


Fig. 3.—Reduction in heart size one year after operation to correct mitral regurgitation.

can predict with remarkable accuracy the amount of mitral regurgitation.

Ideally, this lesion is best corrected without elective arrest. If there is any significant degree of aortic insufficiency however, there is so much reflux blood that visualization and correction is impossible without arrest. One should be aware of this before operation. The reflux dye study of Braunwald and Morrow[10] is important in the preoperative assessment of aortic insufficiency. Aldridge[11] in our unit has modified this test and has performed it in over 30 cases. When aortic insufficiency is present we have used anoxic arrest to maintain a bloodless field.

A right postero-lateral incision is used and the left atrium is incised posterior to the lung root. The annuloplasty is carried out with Ivalon reinforced stitches. All these patients were in an advanced stage of their disease and most of them had pure insufficiency. Sixteen patients have been operated upon. There were three deaths and one late recurrence in the first five cases. In the last 11 cases there have been no deaths. All have been remarkably improved clinically and all have maintained their improvement. Fig. 3 shows a one year follow-up chest radiograph with marked reduction in heart size. Fig. 4 illustrates the reflux dye curve at left heart catheterization before and after surgery in another patient.

With proper selection of cases, annuloplasty for mitral regurgitation may prove a very worthwhile operation. Our very encouraging results were probably aided by the use of hypothermia during cardiac bypass.

The Future of Hypothermia

The use of cardiac bypass with deep (10° C. to 20° C.) hypothermia is still

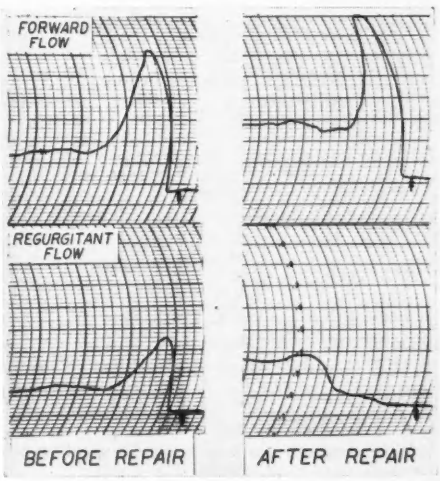


Fig. 4.—There is no evidence of regurgitation in the postoperative dye studies: Simultaneous sampling of dye injected in the left ventricule. Upper panel shows values of forward flow measured by the ear oximeter. Regurgitant flow cuvette in left atrium is seen in the lower panel.

experimental. The studies of Gollan[12] and others have been repeated by Heimbecker[13] who has successfully combined cardiac bypass with a body temperature of 15° C. in dogs, with cardiotomy and survival. All other experimental workers in this field have encountered a high mortality rate.

The reports of Drew[14] are interesting in this regard. He has had clinical success in the use of deep hypothermia with extracorporeal cooling without an oxygenator. There is good evidence that human tolerance to profound hypothermia is similar to that of animals and that the age of the patient is an important factor. Newborn dogs can be cooled to 5° C. with survival.[15] This tolerance to profound hypothermia disappears gradually with maturity and reaches a plateau in the adult. Drew's technique of deep hypothermia is both rational and timely as it is applied to children. One would expect definite limitations in tolerance to low body temperature with less success when applied to a number of adults.

Our research team is still investigating the phenomenon of hibernation[16] with the hope that this knowledge will increase the tolerance of the body to deep hypothermia and perhaps extend the range of safe body temperature to 8° C. or 10° C.

One and one-half hours of intracardiac surgery is currently safe with cardiac bypass, with or without moderate hypothermia. Our goal is to increase the safety of a two to three hour procedure and produce a simple physiological technique of surgery with a minimum of cannulæ and apparatus.

Summary

Refinements in the technique of hypothermia are discussed.

The surgical correction of atrial septal defects is reviewed as an example of the use of simple hypothermia.

The clinical use and experimental status of hypothermia combined with cardiac bypass is assessed. As an example of the clinical application of this combined technique, the open heart surgical treatment of mitral regurgitation is reported.

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Résumé

L'hypothermie clinique variant de 28° C. à 31° C. est devenue de toute sécurité, en autant que le contrôle de la température corporelle soit maintenu, que l'irritabilité cardiaque soit réduite au minimum, que le "shock" par réchauffement soit éliminé, et que la tendance occasionnelle à l'hémorragie, surtout chez les adultes, soit annihilée.

L'exclusion cardiaque associée à l'hypothermie profonde (10° C. à 20° C.) demeure encore au stage expérimental. Les expériences de Gollan ont été reprises par Heimbecker qui a combiné, avec succès, l'exclusion cardiaque avec une température de 15° C., chez le chien, pour exécuter les cardiotomies avec survie. Tous les autres expérimentateurs, ont affronté un taux de mortalité élevé.

Drew a rapporté des succès cliniques en se servant d'hypothermie par refroidissement extra-corporel, sans oxyginateur. La tolérance humaine à l'hypothermie profonde semble superposable à celle des chiens et l'âge devient un facteur important. Les chiens nouveau-nés peuvent être refroidis à 5° C. et survivre, mais cette tolérance s'atténue progressivement, avec la maturité, pour demeurer en plateau chez l'adulte. La technique de Drew est aussi rationnelle qu'à point, puisqu'appliquée aux enfants.

Notre équipe expérimentale étudie encore le phénomène d'hibernation, dans l'espoir d'augmenter la tolérance corporelle à l'hypothermie profonde.

La chirurgie intra-cardiaque qui dure une heure et demie est actuellement sans danger, avec ou sans exclusion cardiaque, avec ou sans hypothermie modérée. Notre but est d'accroître la sécurité des opérations qui durent deux à trois heures et de produire une technique simple qui n'exigera qu'un minimum de cannules et de montage.

TYPICAL GYNECOLOGIC OPERATIONS. Dr. Siegfried Tapfer, in charge of the University Clinic for Gynecology and Obstetrics, Innsbruck, Tirol, Austria. Translated by L. M. Szamek, M.D., New York. 81 pp. Illust. 2nd ed. J. P. Lippincott Company, Philadelphia and Montreal, 1960. $9.00.

This book does not set out to be a textbook, and it is not one. It shows the art of surgery as practised by many well known and respected continental surgeons.

The illustrations are very clear and show each step of the procedures with the text following the pictures very well.

Many of the procedures described are not done by British or American surgeons nowadays. The radical surgery described is not as radical as usually performed for carcinoma. In the text they explain that their results warrant this less extensive surgery, but few figures are quoted and none are up to date, so that this statement is hard to verify.

The book makes pleasant reading for those interested in the art of surgery, and also from a historical viewpoint.

THE TRIUMPH OF SURGERY. Jurgen Thorwald. Translated by R. and C. Winston. 454 pp. Illust. Pantheon Books Inc., New York; McClelland and Stewart Limited, Toronto, 1960. $7.00.

What has happened in surgery in the past century is a wonderful story and medical men know it. But doctors are scientists and they write the history of this era critically, with facts and dates and logic, with little expression of the fantastic daring, of the emotions involved and of the rivalries, personalities and feuds. *The Triumph of Surgery* tells of those surgical giants who first dared the operations that are now so common: thyroidectomy, cord tumour, cholecystectomy, herniorrhaphy, pneumothorax, corneal transplant, craniotomy, spinal anæsthesia.

Some may find the telling of these stories by an imaginary surgeon traveller apocryphal. Historical personalities are described and quoted as are the characters in a novel. There is evidence of a great deal of research into the personalities of the founders of modern surgery and the background of each "breakthrough". The stories are told well and the great occasions might have been as tense and emotional as this. These "firsts" are so historic that they should be written for all to read.

The Triumph of Surgery is especially suitable for the layman, the medical student and the doctor who does not make a hobby of the history of his profession.

MODERN TRENDS IN ACCIDENT SURGERY AND MEDICINE. Edited by Ruscoe Clarke, M.B.E., M.B., F.R.C.S.(Eng.), F. G. Badger, B.Sc., F.R.C.S. (E), and Simon Sevitt, M.D., M.Sc., F.R.C.P.I., D.P.H., of the Birmingham Accident Hospital, England. Butterworth & Company (Toronto) Limited, 1959. $15.00.

This is much more than another textbook on emergency surgery for it consists of a series of discussions on various aspects of trauma by members of the staff of the famous Birmingham Accident Hospital. The patient as a whole and the broad aspects of his situation as an accident victim and a surgical problem is the theme. Medical, physiological and pathological viewpoints are emphasized. The volume is kept reasonable in size by leaving out several large subjects: e.g. burns, anæsthesia and head injuries.

Surgeons who deal with trauma, orthopædic, plastic, or general surgery, will find this book stimulating. From the first chapter on the organization of accident services, through blood transfusion, uræmia, technique of wound surgery, fractures, embolism, hand, face and nerve injuries to rehabilitation, the writing is clear, thoughtful, and up to date.

The book is beautifullv printed, illustrated and bound. Surgeons and students of surgery will find it a valuable addition to their libraries.

EXAMINATION OF THE BILIARY TRACT WITH CHOLOGRAFIN: A SURGICAL-RADIOLOGICAL CORRELATION*

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THIS REPORT is based on a detailed analysis of 597 examinations of the biliary tract with intravenous Cholografin, in 216 of which, a surgical-radiological correlation was possible. It includes the findings of our first series of 228 cases which were presented at the meeting of the Royal College of Physicians and Surgeons of Canada at Toronto in October 1956, and which were subsequently published.[1]

PHYSIOLOGY

In the presence of good liver function, 90% of the injected Cholografin is excreted by the liver, causing rapid opacification of the bile ducts if the medium is able to collect therein.[4] Cholografin is not concentrated by the gallbladder, but if the cystic duct is open, and if the medium is able to pool in the gallbladder, its density allows fairly rapid opacification of this organ but does not give information regarding gallbladder function.[2, 12] It is reported that Cholografin does not enter the enterohepatic circulation as does Telopaque and certain other oral preparations. If such be the case it should not cause reopacification of the biliary tract after it has reached the bowel, but there is some controversy regarding this statement.[10]

PREPARATION OF THE PATIENT

Our procedure regarding the important consideration of proper preparation of the patient has been as follows: A thorough cleansing of the bowel with castor oil and a total fast from all food and drink for 12 hours before the examination, was carried out. No drug was used to disperse troublesome gas when it was encountered.

SELECTIONS OF PATIENTS

Some of the patients had had a previous examination using an oral medium (Telopaque) but in others Cholografin was used in the initial examination. The patients were examined on both an in hospital and outpatient basis and were selected for such examination by their physicians. No particular age group was deliberately excluded but the series did not include any children or any cases following acute trauma.[7, 9]

ROUTINE OF EXAMINATION

The manufacturer's suggested routine of sensitivity testing, administration and radiography was modified somewhat. Preliminary conjunctival instillation was omitted, but the intradermal test was employed, as was an intravenous test dose of 1 c.c. of the medium. Following this 40 c.c. of 20% solution of the sodium salt, or later in the series 20 c.c. of the methylglucamine salt was injected routinely. Serial radiographs were made at five, 10, 15 and 30 minute intervals, with additional films at one, two and three hours, or at such other intervals as the radiologist considered to be indicated. The radiologist was in continuous and active supervision of the examination, making changes in exposures, technique and positioning as he considered necessary in order to obtain the maximum information. Planograms and fluoroscopic "spot films" of the biliary tract were not made. No drug, such as morphine, that would interfere with the normal physiology of the patient was used, except in the case of certain patients selected for reexamination.[5, 11]

Further modifications in this routine have been introduced after reviewing the films reported in the present series and will be discussed later. In the second series of 368 examinations, a sample of blood for serum bilirubin estimation was taken immediately before the examination.

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*Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, January 1960.

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General Findings

No severe reactions were encountered and "anti allergy" drugs were used only occasionally when minor reactions such as urticaria were encountered. These minor reactions were very few in number and included some nausea and vomiting which were satisfactorily minimized by slow injection of the medium. The injection was usually given over an interval of approximately 10 minutes.[6,8] In these examinations comparison was not made between the size of the bile ducts in those patients who had symptoms of biliary tract disease and those with no symptoms.[3]

The radiologists' impression of the size of the ducts proved to be accurate when compared with T tube or cystic duct operative cholangiograms, or with the operating surgeons' inspection.

There was considerable variation in the size of the common bile ducts in those patients from whom the gallbladder had been previously removed and likewise in those on whom no previous operation had been performed. It is probably not valid to use the size of the ducts alone as an indication of their obstruction or abnormality. It has been stated that in those cases in which the gallbladder has been removed or the cystic duct occluded (i.e. those with no overflow reservoir for the common bile duct), the normal sized ducts varied from 3 mm. to 8 mm. in greatest diameter; that those ducts of 8 mm. to 15 mm. diameter might or might not be involved by stricture or obstruction at the distal end, and that those greater than 15 mm. in diameter were likely to be obstructed to a varying degree.[5,6] We did not find any evidence contrary to this concept.

The quality of visualization of the biliary system throughout the series was considered to be very satisfactory, and with very few exceptions the radiologist was able to make a definite statement as to the presence or absence of calculi.

Detailed Findings

In this study the radiological interpretation of normality does not exclude the possible existence of cholecystitis.[2] Regarding the ducts, it refers to normality in their size and the absence of calculi. This report is divided into two sections. The first, section A, deals with 216 patients whose diagnosis was proven, including the 100 patients in the initial series. The second, section B, concerns the remaining 318 patients whose diagnosis was unproved. Each section will be discussed under the following headings:

Group 1. Cases in which the gallbladder had been previously removed.

Group 2. Cases in which the gallbladder was not visualized.

Group 3. Cases in which the gallbladder appeared normal.

Group 4. Cases in which the gallbladder contained calculi.

Section A. Surgical-Radiological Correlation. (216 Cases with Proven Diagnosis, Table I)

Group 1. 25 patients whose gallbladder had been removed (Fig. 1.)

No opaque medium was seen in the common bile ducts in five of these 25 patients, three of whom had common bile duct obstruction due to calculi with or without associated stricture, and two of whom had fistulae draining the common bile ducts apparently preventing pooling of the opaque medium in the ducts. In the remaining 20 the radiological findings were confirmed at operation. In this group of 25 there were 13 with stones in the common bile duct, three with common bile duct stricture and four with cystic duct stumps.

Group 2. 116 patients whose gallbladder was not visualized (Fig. 2).

In 30 of these cases no opaque medium was visualized in the ducts. Twenty-one of these had sufficient disease of the biliary tract or liver parenchyma to prevent excretion of the medium by the liver in sufficient concentration to opacify the ducts;[3] four showed opaque medium in the duodenum but none in the ducts, indicating lack of adequate contraction of the sphincter of Oddi,[4] and in the other five the same phenomenon must also be postulated since there was neither oper-

TABLE I.—SECTION A. 216 PROVEN CASES

GROUP 1.			
Gallbladder removed 25	(a) Common bile duct not seen		
	(i) Common bile duct obstruction with stones with or without stricture	3	
	(ii) Common bile duct fistula	2	
	(b) Common bile duct with stones		1
	(c) Common bile duct with stricture (no stones)		
	(d) Cystic duct stumps		
GROUP 2.			
Gallbladder not seen 116	(a) Common bile duct not seen		3
	(i) Biliary tract or liver parenchyma disease	21	
	(ii) Opaque medium in duodenum only	4	9
	(iii) Cause unknown	5	
	(b) Opaque medium excreted by liver		9+86=9
GROUP 3.			
Gallbladder normal 14	(a) Gallbladder with calculi 3, Gallbladder normal 11		
	(b) Common bile duct with calculi		
	(c) Common bile duct with stricture, no calculi		
	œdema head of pancreas		1
	(d) Common bile duct enlarged carcinoma head of pancreas		1
GROUP 4.			
Gallbladder with calculi 61	(a) Gallbladder with calculi		6
	(b) Common bile duct with calculi		12

ative, clinical nor laboratory evidence to explain lack of excretion by the liver.

In the 95 cases in which the liver apparently excreted opaque medium, non visualization of the gallbladder was due to either obstruction at or in the cystic duct or neck of the gallbladder by calculus, fibrosis, and/or œdema, or to a fistula from the gallbladder preventing pooling of the media within it.

There were two instances of radiological misinterpretation. In one case a calculus was missed in the radiographic investigation, and in another, the presence of a calculus was suspected but not confirmed at operation. In the remainder, the radiological findings were confirmed at operation.

Group 3. 14 patients whose gallbladder appeared normal (Fig. 3).

In three of these patients opacification of the gallbladder was poor, and calculi later proven to be present were not recognized on the radiographs. In addition, a calculus was reported in a dilated duct in one patient but its presence was not confirmed at operation. In the remainder, the



Fig. 1.—Post cholecystectomy. Radiograph shows stones in the common bile duct.

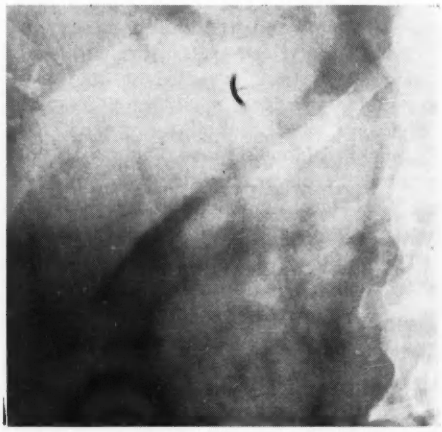


Fig. 2.—Gallbladder not visualized because o stone impacted in neck of gallbladder. Commo bile duct enlarged due to hypertrophied sphincte (1 cm. in diameter). Air outlining biliary tract du to a fistula.

roentgenological findings in regard to both the gallbladder and the common bile ducts were confirmed.

Three of these 14 patients had calculi in their common bile ducts without calculi in the gallbladder; there was a stricture at the sphincter in three others, and the distal end of an enlarged duct was involved by œdema of the head of pancreas and by carcinoma of the head of pancreas respectively, in two others.

Group 4. 61 patients whose gallbladder contained calculi (Fig. 4).

Calculi were found in the gallbladder at operation in all of these cases. In two instances, although the roentgenograms indicated calculi in the common bile ducts, the surgeon did not find a calculus in one, and in the other, calculi were not detected by palpation, although the duct was not explored. The latter case has been coded as a radiological error. In two other instances, calculi were found at operation, but had not been noted in the roentgenograms. As there had been about a three month interval between radiographic examination and operation, it is possible that these calculi had passed into the ducts from the gallbladder during the interval. However these two cases have also been designated as radiological errors.

There were two cases of unusual interest in this group. The first was one in which the common bile duct was not visualized. At operation this duct was found to be enlarged and literally packed with small calculi and "biliary mud", and in addition an operative cholangiogram disclosed an aberrant right hepatic duct which also contained multiple calculi. These could not be removed and have since been the cause of repeated bouts of colic. The second case concerns that of a patient with three calculi found at operation in a normal sized duct by a routine cystic duct cholangiogram. As indicated above, the radiologist did not see these calculi (Fig. 4), nor could the surgeon palpate them even though he knew that they were present.

Accuracy of Roentgenological Interpretation

In these 216 proven cases there were 10 errors, an error of 4.6% or accuracy of 95.4% from this investigative technique. As has been indicated, the roentgenological interpretation in three of these 10 cases might have been correct at the time the films were made. Of the 10 errors that were coded, seven were in the first 100 cases examined and only three in the second group of 116.

All these facts indicate that if the radiologist is familiar with this form of examination, and that if it is carefully performed and supervised, its accuracy is of a very high degree.



Fig. 3.—Gallbladder normal. Common bile duct containing stones, and enlarged due to œdema of head of pancreas.

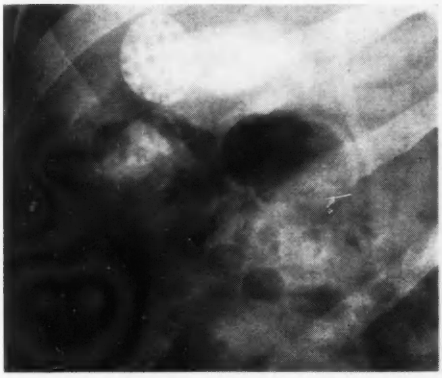


Fig. 4.—Gallbladder containing calculi. Stones present in common bile duct but not visualized by radiologist.

TABLE II.—Section A. Non Opacification of the Bile Ducts. 216 Proven Cases

1.	Biliary tract or liver parenchyma disease		24
	Impacted calculus (a) Carcinoma head of pancreas } with dilated common bile duct Œdema head of pancreas		
	(b) Cirrhosis of liver Secondary carcinoma of liver Abscess of liver Ascending cholangitis		
	(c) Clearing or clinical jaundice		
2.	No known reason	5	13
3.	Common bile duct fistula	2	
4.	Common bile duct packed with calculi and "biliary mud"	1	
5.	Inadequate contraction of sphincter of Oddi	5	

Non Opacification of the Bile Ducts of 216 Proven Cases. Table II.

In 24 of the 216 cases in which the diagnosis was proven, there was either clinical, laboratory or operative evidence of biliary tract or liver parenchyma disease of sufficient magnitude to explain lack of excretion of the opaque medium by the liver, and hence non opacification of the biliary tract. These disorders included one or more of the following (a) dilated common bile duct associated with impacted calculi and/or œdema of the head of pancreas, or carcinoma at the distal end of the duct; (b) liver disease due to cirrhosis, extensive secondary carcinoma, liver abscess or ascending cholangitis, or (c) clinical or clearing jaundice.[3]

In 13 cases, there were no reasons from the operative, clinical or laboratory findings to explain why the opaque medium was not excreted and it is presumed that it actually was excreted. We have no reasons to explain lack of opacification of the ducts in five of these patients. In two common bile duct fistulae were present which would account for such lack of visualization; in one, the enlarged duct was completely packed with calculi and "biliary mud", and in the other five there was evidence of lack of adequate contraction of the sphincter of Oddi. This evidence consists of opaque medium visualized in the duodenum but none in the biliary tree and/or visualized gallbladder without visualization of the ducts, or a successful repeat examination using morphine sulphate to augment contraction of the sphincter of Oddi.[4]

Section B. The Unproven Cases (381 Examinations, Table III)

Group 1. 114 patients whose gallbladder had been removed

The bile ducts were not seen in eight

TABLE III.—Section B. 381 Unproven Cases

GROUP 1. Gallbladder removed 114	(a) Common bile duct not seen			8
	(i) Cause unknown	1		
	(ii) Biliary tract or Liver disease	7		
	(b) Common bile duct with calculi	6		
GROUP 2. Gallbladder not seen 80	(a) Common bile duct not seen			23
	(i) Biliary tract or liver disease	13		
	(ii) Medium in duodenum	1	Inadequate contraction sphincter of Oddi	10
	(iii) Repeat with morphine	3		
	(iv) Unknown	6		
	(b) Common bile duct with calculi			3
GROUP 3. Gallbladder normal 138	(a) Common bile duct not seen (Inadequate contraction sphincter of Oddi)			2
	(b) Common bile duct with calculi			2
GROUP 4. Gallbladder abnormal 49	(a) Polyp 1, calculi 48			
	(b) Common bile duct not seen (Inadequate contraction sphincter of Oddi)			4
	(c) Common bile duct with calculi			2

of these cases. There was no apparent reason to explain the non opacification of the ducts in one case, but in the other seven there was either laboratory or clinical evidence of biliary tract or liver parenchyma disease (recurrent cholangitis, clinical jaundice or elevated serum bilirubin).[3] In six cases the common bile ducts were considered to contain calculi but to date we have no records of operation in these patients.

Group 2. 80 patients whose gallbladder was not visualized

In 23 of these patients the common bile duct was not seen. There was either clinical or laboratory evidence of liver parenchyma or biliary tract disease in 13 to explain lack of secretion by the liver and hence non opacification of the biliary tract. This evidence included clinical jaundice, known obstruction of the common bile duct, extensive carcinoma of the liver, acute cholecystitis with either cholangitis or pancreatitis, elevation of serum bilirubin, or subsiding jaundice.[3] In one of the 23 cases, opaque medium was present in the duodenum, and in the other nine no reason for lack of its excretion by the liver was apparent from the records. However, since a repeat examination using morphine sulphate to augment contraction of the sphincter of Oddi was successful in demonstrating the ducts in three of these, we are probably permitted to postulate lack of contraction of the sphincter of Oddi in these nine patients.[4]

Only three of these 80 cases showed calculi in the ducts.

Group 3. 138 patients whose gallbladder appeared normal

As the gallbladder was visualized in all of the patients in this group and as the common bile ducts were not opacified in two of them, it was presumed that in these two instances the sphincter of Oddi did not offer sufficient resistance to prevent rapid passage of the opaque medium into the duodenum.[4] Only two patients in this group showed calculi in the common bile ducts.

Group 4. 49 patients whose gallbladder appeared normal

The presence of a polyp was suggested in the gallbladder in one of these cases, and calculi in the remainder. In four the ducts were not seen, suggesting inadequate contraction of the sphincter of Oddi as in group 3, above.[4] The presence of calculi in the common bile ducts was suggested in 2 cases. Some of the patients in this group may have been operated upon elsewhere or may be awaiting operation at a later date.

NON OPACIFICATION OF THE BILE DUCTS OF 381 UNPROVEN CASES, TABLE IV

In 20 of these 381 examinations there was either clinical or laboratory evidence of biliary tract or liver parenchyma disease of sufficient magnitude to explain lack of excretion of the opaque medium by the liver, and hence non opacification of the biliary system. These included recurring cholangitis, clinical or clearing jaundice, known obstruction of the common bile duct, extensive carcinoma of the liver, infectious hepatitis, acute cholecystitis, with cholangitis or pancreatitis or elevation of serum bilirubin.[3]

In 10 patients there was good evidence of inadequate contraction of the sphincter of Oddi as indicated in Section A, and in seven additional cases the reasons for non opacification of the ducts were not apparent, but in these the liver probably excreted the opaque medium nevertheless.[4]

TABLE IV.—SECTION B. NON OPACIFICATION OF BILE DUCTS. 381 UNPROVEN CASES

1. Biliary tract or liver parenchyma disease	20
(a) Recurring cholangitis	
(b) Clearing or clinical jaundice	
(c) Known obstructed common bile duct	
(d) Extensive carcinoma of liver	
(e) Infectious hepatitis	
(f) Acute cholecystitis with cholangitis or pancreatitis	
(g) Elevated serum bilirubin value	
2. Inadequate contraction of sphincter of Oddi	10
3. No known reason	7

General Analysis (Sections A and B)

Comparison of results from intravenous Cholografin with those of orally administered media.

In 100 of the 597 cases the examination with intravenous Cholografin was successful in visualizing at least part of the biliary tree, following failure of the oral medium (Telopaque) to do so. In 37 of these cases both the gallbladder and the bile ducts were seen, and in 63 cases only the bile ducts were seen. This would indicate the value of Cholografin in attempting to arrive at a correct preoperative diagnosis.

Relationship of serum bilirubin values to opacification of the biliary tract (normal value less than 1.0 mg.%).

Serum bilirubin values were recorded in association with 365 examinations. No opacification of the biliary system occurred in the presence of a serum bilirubin level greater than 1.8 mg. %. In only four cases with serum bilirubin greater than 1.5 mg. % was the biliary system visualized. This critical level is considerably lower than that suggested by other reports.[5,6,12]

A normal serum bilirubin level was not always associated with opacification of the biliary tract. The reason for this is readily apparent when one considers that the bilirubin level is only one index of liver function and that it may have returned to normal while other tests indicated continuing impairment of liver function.

In this series there is no apparent relationship between the size of the ducts and the rapidity or optimum degree of their opacification.

Non Opacification of the Biliary Tract (597 Examinations)

There were no obvious reasons to explain the failure of visualization of some part of the biliary tree in 12 of the 597 examinations (about 2%). This figure might have been further reduced if we had had more complete clinical information, or if these examinations had been repeated using morphine sulphate.

Fifteen, or 2.5% of the total showed definite evidence of lack of adequate contraction of the sphincter of Oddi.[4]

There were two known common bil duct fistulae, and one duct filled wit "biliary mud" and calculi.

In 44, or 7.3% there was sufficient biliar tract or liver parenchyma disease to pre vent excretion of the opaque medium.[3]

These figures suggest that if one wer to exclude those cases in which visualiza tion could not be reasonably expected and if the radiologist were to supervis carefully each examination, and repeat th examination in selected cases using mo phine sulphate, a successful examinatio could be expected in a high percentag of cases.

In this series of 597 examinations, eve including those cases in which opacifica tion would not be anticipated, some par of the biliary system was seen in 89%.

Modifications in Radiographic Technique

The radiographs were reviewed with reference to the initial and the optimum visualization of the ducts and gallbladder. Many of the films which had been taken were found to be unnecessary. In most cases the ducts were seen best on the 30 to 60 minute films. As a result, the routine of radiography has been revised. We now take a scout film, and then take P.A. and oblique films at 10 minute, 30 minute, one hour, two hour and four hour intervals, and discontinue the examination as soon as the necessary information has been obtained. Each film is examined by the radiologist after immediate processing, and any necessary change in technique or positioning is ordered. The use of morphine is limited to repeat examinations, in cases in which the first attempt has not been successful and in which the liver is believed to be capable of excreting the medium. The exposures used for an average patient were 65 K.V. and 150 M.A.S. using cassettes with high speed screens and Potter-Bucky grid (12:1).

This new routine has proved satisfactory, has reduced radiation exposure to the patient, and has allowed the radiographic equipment to be used in the interval between films for other purposes.

Suggested Reasons For Non Excretion of Opaque Medium by the Liver

1. Infectious hepatitis.
2. Secondary malignancy of liver.
3. Cholangitis.
4. Liver abscess.
5. Cirrhosis.
6. Obstructive jaundice.
7. Clearing jaundice.
8. Elevated serum bilirubin.

Suggested Reasons For Non Opacification of the Gallbladder

1. Gallbladder previously removed.
2. Obstruction of or at the cystic duct or neck of gallbladder by calculus, fibrosis or œdema.
3. Fistula from the gallbladder (preventing pooling of the opaque medium).
4. Very small fibrosed gallbladder with practically no lumen (following previous cholecystotomy).
5. Lack of adequate contraction of the sphincter of Oddi (allowing the opaque medium to flow freely into the small bowel[4]).

Suggested Reasons For Non Opacification of the Ducts

1. Duct packed with calculi and/or "biliary mud".
2. Fistula from the duct or previous anatomical interruption of the duct.
3. Lack of adequate contraction of the sphincter of Oddi.[4]

Conclusions

The use of intravenous Cholografin is a valuable and accurate method of examining the biliary system but it should not replace a careful clinical evaluation.

We reaffirm the belief stated in our previous report, that the initial method of radiological examination should be by means of an orally administered medium.

Intravenous examination should be used if the gallbladder has been previously removed or if the desired information regarding the gallbladder or ducts has not been obtained using the oral method.[2]

Errors in interpretation are more likely to occur if opacification of the biliary system is poor.

Examination was not successful in the presence of clinical jaundice or in cases with extensive biliary tract or liver parenchyma disease.[3]

No opacification of the biliary tract occurred if the serum bilirubin value was greater than 1.8 mg. % (and was poor at this level), but a normal serum bilirubin value did not ensure that the examination would be successful.

In selected cases it may be necessary to augment tone of the sphincter of Oddi by the use of a drug such as morphine.

Reasons for non excretion of the Cholografin by the liver, and for non opacification of the gallbladder and biliary ducts have been suggested.

The routine of the examination has been revised, reducing the number of exposures.

Some part of the biliary tract was successfully visualized in 89% of cases, and this figure might have been further improved by more rigid selection of cases and careful supervision of the examination by the radiologist.

No serious reactions to Cholografin were encountered in this series of 597 examinations. Minor reactions such as nausea, vomiting and urticaria were easily controlled by injecting the medium slowly and occasionally by the added use of an "anti allergy" drug.

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Résumé

L'utilisation du Cholografin intra-veineux constitue un moyen d'examen de l'arbre biliaire utile et précis, mais ne devrait jamais remplacer l'évaluation clinique minutieuse.

Cet examen ne devrait être employé que si la vésicule biliaire a déjà été enlevée ou si les renseignements désirés n'ont pu être obtenus par la méthode d'opacification orale. Les erreurs d'interprétation surviendront surtout lorsque l'opacification sera faible.

Dans les cas de jaunisse ou d'atteinte parenchymateuse ou biliaire étendue, l'examen n'a pas été révélateur.

Avec une bilirubine sérique dépassant 1.8 mg %, l'opacification ne s'est pas produite, quoique sa valeur normale ne soit pas un critère d'opacification certaine. Dans certains cas, il a été nécessaire d'augmenter la tension intra-cholédocienne en se servant de Morphine.

Quelque portion de l'arbre biliaire s'est effectivement visualisée dans 85% des cas; une sélection plus rigide associée à la surveillance étroite du protocole de l'examen par le radiologiste devrait encore donner un meilleur pourcentage.

Aucune complication sérieuse ne s'est manifestée au cours de ces 597 examens. Des réactions sous forme de nausées, de vomissements et d'urticaire étaient contrôlées par l'injection ralentie du médium opaque et par l'addition occasionnelle d'une médication anti-allergique.

CHIRURGIE DE L'ETAGE SUPERIEUR DE L'ABDOMEN. Tactiques techniques opératoires régionales et anatomie évolutive. Henri Fruchaud avec la collaboration de A. Bernou, A.-F. Lemanissier, G. Lessertisseur et G. Videau. 198 pp. Illust. G. Doin et Cie, Paris, 1960. 32NF.

Il y a deux parties à ce livre: la première traitant de l'Anatomie évolutive, la seconde de tactiques et techniques opératoires.

La première partie est intéressante et nous donne une description probablement juste de l'évolution dans la disposition des organes chez l'être humain quand il a adopté la posture redressée.

On comprend bien comment la cage thoracique s'est rétrécie et comment a leur tour les organes abdominaux supérieurs se sont tassés sous un diaphragme qui a acquis chez l'humain une forme différente de celle de l'animal.

Des dessins bien faits nous expriment bien la formation des bandes péritonéales et les moyens de soutien des organes de l'étage supérieur.

La seconde partie démontre l'agrandissement considérable du champ opératoire obtenu par la position du malade sur la table et surtout par l'emploi d'écarteurs orthostatiques qui une fois fixés donnent un jour qui paraît supérieur à tout autre.

Le seul inconvénient semble-t-il est que le colon transverse et son méso et le grand épiploon sont extériorisés et ont perdu droit de cité dans l'abdomen.

Cet état cependant facilite les mouvements de traction qu'il faut exercer au cours des manœuvres opératoires sur les organes situés dans les hypochondres.

Dans les techniques opératoires décrites on insiste surtout sur les avantages anatomiques obtenus par la section des bandes péritonéales qui permettent une très grande mobilisation viscérale.

Le livre est intéressant.

Les tactiques chirurgicales méritent d'être relues.

SURGICAL GASTROENTEROLOGY. Considerations based on Pathologic Physiology. Warner F. Bowers, A.B., B.Sc., M.D., M.Sc., Ph.D. (Surg.), Diplomate of the American Board of Surgery, Diplomate of the National Board of Medical Examiners. 498 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $22.25.

The author has called upon his extensive experience, particularly in the military field, to present a most interesting approach to surgery of the gastrointestinal system. He outlines, under special diagnostic procedures, a positive wealth of information of particular value to the resident surgeon engaged in such clinical evaluations as "diagnostic biliary drainage", "liver biopsy" and "gastroscopy". Preoperative and postoperative care are considered. It would be difficult to discover any congenital or acquired lesion from œsophagus to anus that is not discussed from the point of view of pathological physiology. An attempt is made to explore the reasons why surgery should be considered for a specific condition and to answer the question as to what may be expected from operation.

This volume, which is apparently unique in the field makes a very valuable addition to the library of the gastrointestinal surgeon.

AN ANALYSIS OF OPEN AND CLOSED TREATMENT OF FRACTURES OF THE TIBIAL SHAFT*

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FRACTURES OF THE SHAFT of the tibia have continued to present a problem in management. Although closed reduction and maintenance of position by a cast produces satisfactory results in a large proportion of such fractures, there is still considerable disagreement in respect to methods of internal fixation and advantages and disadvantages of this type of treatment. It has been stated categorically that open reduction inevitably leads to prolongation of healing time, as well as producing a tendency toward other complications, particularly infection. For these reasons, many have adopted an extreme point of view in avoiding open reduction and internal fixation, even in cases which are not well managed by closed techniques.

In an attempt to clarify the relative status of open and closed management of fractures of the tibial shaft, all such cases treated at this hospital during the past eight years have been analyzed with respect to rate of healing and incidence of complications. It must be recognized that such an analysis does not represent a controlled study of various groups with random selection of cases for the respective types of treatment. In general, fractures treated by open methods represent the more severe injuries in respect to degree of comminution and instability. Open reduction has usually been reserved for fractures which were unsuitable for management by closed methods or in which reduction was not maintained by a cast alone. Nevertheless, it was felt that such an analysis would prove useful in determining whether any real disadvantages were apparent with one or other method of treatment.

MATERIAL AND METHOD

A total of 91 fractures of the tibial shaft were treated during the interval studied. The only element of selection of cases for study was in respect to age, patients under 21 years being arbitrarily excluded since fractures in the very young ordinarily present lesser problems in management. In the majority of these cases treatment was carried out by the resident staff, whether by open or closed methods, and the nature of the fracture was not a determining factor in whether the case was managed by the house staff or an attending physician.

RESULTS

Closed Treatment

Twenty-seven patients with simple fractures of the tibial shaft had initial closed reduction and application of casts. In four of these patients a satisfactory position was not maintained and subsequent open reduction‡ was necessary. The remaining 23 patients obtained satisfactory healing without complications.

Twenty-three patients with comminuted fractures had an initial attempt at closed reduction. Seven of these required subsequent open reduction because of unsatisfactory position and the remaining 16 achieved primary union.

Six patients, four with simple fractures, and two with comminuted fractures, were treated by closed reduction with transfixion pins above and below the fracture site incorporated in the plaster cast. Of these, one patient had a delayed union and one patient had non union requiring secondary open reduction and bone graft.

Open Reduction

Twenty-six patients had open reduction and internal fixation by transfixion screws. All of these achieved primary healing except one in whom delayed union occurred, healing in 11 months. In this single case incorrect use of a transfixion screw

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‡Internal fixation was applied in all cases in this series in which open reduction was performed.

TABLE I.—AVERAGE HEALING TIME

	Type of fracture	*Type of treatment*	*Healing time Weeks ± S.D.*
A.	Simple	Closed	14.9 ± 4.6
B.	Simple	Open	16.3 ± 5.0
C.	Comminuted	Closed	18.9 ± 6.8
D.	Comminuted	Open	17.7 ± 5.9

Significance of differences in healing times

A. vs B.:	Not significant	t = .91	.40 P .20
C. vs D.:	Not significant	t = .59	.60 P .40
A. vs C.:	Significant	t = 2.14	.05 P .01
B. vs D.:	Not significant	t = .89	.40 P .20

resulted in the fracture ends being held in a slightly separated position.

Of six patients in whom a bone plate was used, two developed osteomyelitis which responded to treatment and two had delayed union which responded well to secondary bone grafts.

Three patients with primary bone grafts obtained primary healing without complication.

The mean time interval from injury to clinical union for the different groups is illustrated in Table I. Healing time was interpreted as the interval from the occurrence of the fracture to the point at which radiographs and physical examination indicated sufficient healing to warrant removal of the cast. For simple fractures the mean interval with closed treatment was 14.9 weeks and with open treatment 16.3 weeks (Table I), a difference which is not statistically significant. For comminuted fractures the mean interval to healing was 18.9 weeks with closed treatment and 17.7 weeks with open treatment. The increased time required for union in the comminuted fractures is statistically significant when compared with the healing time for simple fractures, but the difference in healing time between closed and open treatment of comminuted fractures is not.

In Fig. 1 the individual healing times in the various groups are plotted individually, illustrating the dispersion.

DISCUSSION

Open reduction and internal fixation of fractures of the tibial shaft has been practiced for many years. Because of certain theoretical or actual hazards this method of treatment is ordinarily employed only when there seems little reasonable possibility of achieving a satisfactory result by closed methods. It is difficult to quarrel with this point of view since, at best, any operative procedure involves the risks of anæsthesia and the apparently inevitable although small danger of infection attendant upon any operative incision. With modern anæsthetic techniques, the hazard of infection would appear to be a more significant danger. It is difficult to assess the effect of prophylactic use of antibiotics on the incidence of wound infection in elective surgery; however, there is no doubt that their use in treating infection has lessened the gravity of this complication.

Perhaps a more commonly considered contraindication to open reduction has been the supposed adverse effect on the rate of healing of the fractures. Urist *et al.*[1] in an exhaustive review of cases of delayed union and non union concluded that simple fractures healed more rapidly than comminuted ones and that compound (open) fractures did not show a prolonged healing time; these conclusions are supported by the present study. However, they also expressed the view that "comminuted fractures of the human adult tibia should be considered non operable fractures during the first six months of healing because the trauma added by surgery exceeds the normal capacity for bone regeneration in this area of the skeleton". This present study does not support the contention that open reduc-

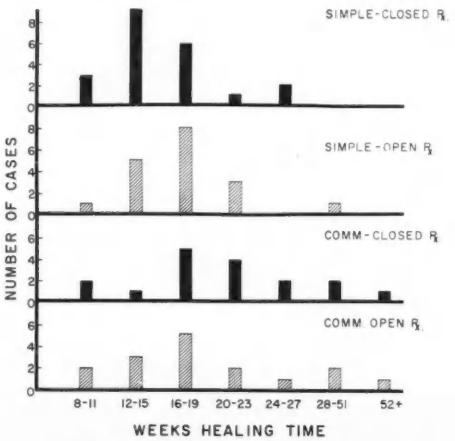


Fig. 1.—Distribution of healing times.

ion *per se* delays the rate of fracture healing. When similar groups of patients are compared, it is seen that the average rate of healing is not significantly different in the groups treated by open reduction and internal fixation from those treated by closed reduction. Therefore, the probability of delayed healing does not appear to be a valid contraindication. It is possible that the previous assumption in this respect has been due to the fact that noncomparable groups of patients were studied and that factors other than the open reduction itself were the determining ones in the delay of healing. The healing time in this series agrees rather well with the studies of Jackson and Macnab[2] who found an average healing time of 17.2 weeks in simple fractures and 21.1 weeks in comminuted fractures treated by closed methods.

Although the development of the slotted plate and its proper utilization has been an improvement, the natural tendency of a plate is to hold the fracture ends in a fixed longitudinal relationship so that resorption of bone at the fracture ends tends to produce a gap between the fragments which will delay healing. This effect also occurs with multiple pin fixation when held in a permanent position such as with plaster, as was seen in this series. This unfavourable effect should not obtain in fractures which are oblique or spiral and can be treated with single or multiple screw fixation. The use of a sliding or onlay bone graft, which may also tend to hold the fracture site separated, produces a compensatory beneficial effect on healing which would appear to offset this disadvantage.

These theoretical considerations are well substantiated by this series and others[2, 3] since uniformly good results were obtained in patients subjected to multiple screw fixation of oblique or spiral fractures or primary bone graft. The early removal of transfixion pins, allowing the fracture ends to move into longitudinal apposition should lessen the complication rate with this type of management.

Although the overall incidence of complications was appreciably higher in patients treated by open reduction, a more detailed inspection of this group of patients revealed that most of the poor results were attributable to a single type of internal fixation, namely the use of a bone plate. Similar difficulties with this method of internal fixation have been reported by others. In assessing the relative merits of various methods of management, the competence of the physician in a particular method of therapy may well be a determining factor and it is possible that this factor may have influenced the results achieved with bone plates in this and other series.

Although primary open reductions frequently have been carried out with success in open fractures, one should expect the risk of infection to be significantly higher in this group and it would appear desirable to obtain primary closure of the wound, with secondary open reduction and internal fixation, when necessary, being carried out after wound healing is accomplished.

Summary

A consecutive series of 91 fractures of the shaft of the tibia has been analyzed with respect to rate of healing and incidence of complications by open and closed methods of treatment. There was a significantly prolonged healing time in simple fractures as compared to comminuted fractures.

Open reduction and internal fixation did not significantly prolong the time of healing over that obtained with closed reduction in comparable types of fractures.

A somewhat higher incidence of complications was observed following open reduction. These complications occurred almost exclusively in those fractures treated by a bone plate which tended to maintain a fixed longitudinal relationship of the fracture ends. Similar difficulties were observed in fractures treated by closed reduction with transfixion pins incorporated into a cast.

It is suggested that when carried out in optimum fashion, internal reduction may be performed without any inherent disadvantage in relation to rate of bone healing and that the incidence of complications should be low.

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RÉSUMÉ

Une série de 91 cas de fractures diaphysaires du tibia a été étudiée pour comparer la vitesse de guérison et l'incidence des complications selon le mode de réduction fermée ou ouverte. Les fractures simples ont guéri plus lentement que les fractures "communitives".

Dans les cas de fractures comparables, les deux moyens de réduction ont donné une vitesse de consolidation superposable. Les complications ont été plus fréquentes dans les cas de réduction ouverte. Celles-ci sont survenues presqu'exclusivement dans les cas de plaques qui visaient à immobiliser l'axe longitudinal des extrémités fracturées. Les mêmes échecs furent cependant essuyés dans les cas de réduction fermée, avec broches incorporées dans les plâtres.

Il ressort que, pratiquée dans des conditions idéales, la réduction sanglante soit acceptable, en ce qui concerne la vitesse de guérison, et que les complications dussent être peu fréquentes.

GEOGRAPHY AND MEDICINE*

"A few centuries ago the Black Death took some ten years to travel from the Orient to western Europe; today an influenza outbreak in a remote Chinese town can spread around the world within weeks.

"Geography has always played a vital role in health and disease, as Hippocrates well knew, but in this modern jet age it has become an integral part of world medicine. Pathogens can now circle the globe in less time than it takes some infectious diseases to incubate. Contrariwise, epidemiologic health teams can descend on an infectious area in a matter of hours, before disease has had time to spread. The winged insect carriers are frequently defeated by man-made winged instruments.

"The earliest form of human being probably began to develop a million years ago, long after the main geographic contours of the earth had been formed. There is a good deal of evidence that Central Asia or Africa were the cradles of the most primitive men.

"If these creatures lived in trees with their anthropoid cousins, any upheaval or change of climate that destroyed forests would force humanids to walk on the ground and develop new postures and skills. Example: some anthropologists believe that the Himalayan range was responsible for reducing rainfall to the north, thus causing forests to shrink toward the south and ultimately disappear.

"Similarly, as the changing pattern of vegetation and rainfall transformed verdant plains into deserts, man and beast moved northward and westward eventually entering Europe. The retraction of the icecap around 20,000 years ago changed the nature of the top soil, profoundly affected the course of rivers and transformed the climate over a vast area. This might be called the last geographic upheaval.

"Primitive human communities now had the choice of several locations: mountainous caves, plains, river valleys, seashores. The peculiar flood and ebb pattern of the Nile made it possible for wild grain (millet, barley) to grow in the warm mud during the winter, also produced a natural system of fertilization and irrigation. Along this river and the great rivers Tigris and Euphrates, were founded the earliest civilizations, apparently entirely by geographic chance.

"The ancient riparian cultures gained some control over geographic forces through primitive dams and irrigation canals, but they were vulnerable to serious climatic changes. Repeated floods in the Mesopotamian basin around 4500 B.C. forced the abandonment of some towns; receding flood waters furnished breeding areas for malarial mosquitoes, contaminated waters may have spread dysentery, cholera, typhoid. One early Babylonian visualized a dark dragon lurking in waters left by a flood. In Egypt, droughts and locusts brought famine; the Nile itself transmitted water-borne infections, and early canals harbored the snail vectors of schistosomiasis. . . ."

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*MD OF CANADA, **1**: 36, 1960.

GALLBLADDER DISORDERS IN THE YOUNG*

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THERE HAVE BEEN 500 cases of cholecystitis with or without cholelithiasis in children or adolescents on record in the whole of surgical literature. Yet there are good reasons to believe that the frequency of this disease is even greater than that which may be reduced by the number of reported cases. Consideration of this disease should enter into the differential diagnosis of obscure abdominal ailments of young patients even if only to be excluded.

Hopmans[1] reported an acute cholecystitis in a six year old boy with concomitant right lower lobar pneumonia. In one series reported by Gross,[2] three of the six patients with cholecystitis also suffered from hæmolytic anæmia. Barnes[3] described a case of acute cholecystitis in a four year old child. A cholecystotomy was first performed and the gallbladder was removed 16 months later since the symptoms and signs had returned. Of Glenn's seven young patients seen at the New York Hospital,[4] one had stones. Lary[5] reported an acute non calcareous cholecystitis with associated mesenteric adenitis in a child. Walker[6] reviewed nine cases in this age group taken from a 15 year period at the Great Ormond Street Hospital. Death of a 14 year old girl was described as resulting from a stone in the common duct with obstructive jaundice, secondary acute inflammation of the biliary tract and subsequent staphylococcal peritonitis. The incidence of common duct stone in cases of cholelithiasis in this age group is estimated at about 4%.[7, 8]

As more information is being gathered on the subject, there is less unanimity among the various theories on ætiology. At the turn of the century, all gallstones in the young were thought to be associated with familial acholuric jaundice. Nowadays, congenital hæmolytic anæmia is not considered so important in causing gallstones, nor are the acute systemic diseases such as typhoid, scarlet fever or other similar infections, as they are less frequent and more easily controlled than they ever were in the past. Tesler[9] mentioned a genetic factor in writing about the occurrence of this disorder in identical twins. Hereditary predisposition has its supporters, as does neonatal sepsis. The finding of stones in the fœtus has been linked to intrauterine cholecystitis and erythroblastosis fœtalis. Other ætiological factors may include sickle-cell anæmia in negroes, the rhesus factor and cystic duct obstruction by an enlarged cystic lymph node. In pointing out that gallstones are less frequently encountered in India than they are in Europe or North America, Ansari[10] wondered whether or not the influence of dietetic factors played any part in their formation. Sodeman[11] suggested that absence or dearth of bile salts caused precipitation of cholesterol; Dannenberg[12] observed that agglutinins favoured bile precipitation which could lead to stone formation. Gallstones in children are usually pigmented with cholesterol and bilirubin as organic components, and lack phosphates, bicarbonates, or calcium. Finally, biliary dyskinesia was suggested by Rankin[13] as an ætiological factor.

In 1941, Ladd[14] thought that before puberty, cholelithiasis was more common than cholecystitis. More recently, Ulin[15] and his associates implied that girls are more susceptible than boys. Babbitt[16] postulated that stones were less frequently silent in the young than in adults. Judging from the information reported on this age group in the literature to date, there seems to be only a nebulous relationship between cholecystitis and cholelithiasis.

Which clinical features may lead to the correct diagnosis? The young patient may present with persistent nausea, vomiting, abdominal pain, diarrhœa and jaundice. This last feature may occur without the presence of a stone in the common bile duct. Back and shoulder pain is said to be rare, possibly because the mechanism of referred pain may not be completely developed at this age. Moreover, pain localization is never easy in examining young

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*This paper was written when the author was on the Resident Surgical Staff, St. Paul's Hospital, Vancouver, B.C.

children. Laboratory findings need not be confirmatory. If the patient has passed puberty a serum amylase level determination may be helpful in cases of cholecystitis when concomitant pancreatitis is suspected; in younger children, the test is of little value.[17] Patients have been discharged with a diagnosis of hepatitis only to be readmitted to hospital a few months or a few years later with cholecystitis or cholelithiasis requiring operation. Since the advent of antibiotics, associated infections are not so frequently seen as they were in the past. The physical signs may be perplexing as often as they are indicative. The gallbladder, liver and spleen may or may not be palpable. Radiological investigation should be employed if the clinical diagnosis is in doubt.

Therapy was formerly conservative because of the frequency of associated infections. It was felt that if the latter could be cured, the gallbladder involvement would regress spontaneously. Cholecystectomy is now the treatment of choice; cholecystotomy should only be used as a life saving measure. Gallstones in the presence of hæmolytic anæmia pose a surgical problem; if the stones are quiescent and do not obstruct the duct, splenectomy should be done first. If the reverse condition exists, cholecystectomy with or without choledochostomy is indicated and the spleen should be left untouched until a subsequent intervention. Fifteen years ago, Waugh operated on a young patient with hæmolytic anæmia and gallstones but without jaundice or cholecystitis, removing the spleen first; as the patient was in excellent condition, the surgeon immediately extended his transverse incision to the right, and performed cholecystectomy. Convalescence was uneventful.[18] Thus it is safe to predict that as operative and anæsthetic techniques advance, simultaneous removal of the spleen and gallbladder will, when necessary, become a one-stage procedure.

Case Reports

Case 1.—A girl 12 years of age was transferred from Ocean Falls Hospital to St. Paul's Hospital, Vancouver in October 1959, with a diagnosis of cholecystitis and cholelithiasis. She gave a three year history of attacks of epigastric pain, at first occasional and nocturnal, never related to meals, lasting from one to four hours and usually relieved by vomiting. Later these attacks began to take place immediately after meals; they were only partially relieved by vomiting and not affected by alkalis. She did not report any food intolerance nor was her appetite affected by the attacks. Although she had been constipated for several years her stools had never been pale; her urine had never been dark and she had been perfectly well between bouts of pain. Two weeks before admission to St. Paul's Hospital she had suffered a severe attack of pain with radiation to the right shoulder. Partial relief was obtained several hours later when she vomited three times. Physical examination on the previous admission had revealed a yellow tinge to her skin. A tentative diagnosis of acute cholecystitis was made. Pain subsided after 24 hours but tenderness remained in the epigastric and right subcostal regions for several days. No family history of gallstones or hæmolytic anæmia could be elicited. The child had always been in good health and had begun menstruating at the age of 10 and one-half years.

She was an obese, well developed 12 year old girl, weighing 148 lb. and was in no acute discomfort. She was not jaundiced. No abdominal tenderness could be elicited; the liver, spleen and gallbladder were not palpable.

Laboratory Investigations

On admission, her hæmoglobin level was 95%, and her white blood count was 9050 per c.mm. with a normal differential count. The serum bilirubin level was 9.6 mg. %; serum phosphorus, calcium and alkaline phosphatase were normal and there was no porphyrin in the urine. No mention of spherocytes was made in the report of the microscopic examination of the blood. A cholecystogram was attempted twice but each time the gallbladder failed to concentrate the contrast medium.

A cholecystectomy and an appendectomy were performed four days after admission. The gallbladder was thickened, chronically inflamed and contained about 50 mixed stones, several of which were impacted in the cystic duct.

Postoperative course was uneventful and the patient is now well. The surgeon feels that this girl may have further stones formed in her biliary system in the future.

Case 2.—A 15 year old girl was admitted in early January 1960, complaining of right upper quadrant pain for the previous three

months. She had been admitted once before n St. Paul's Hospital, Vancouver in October 959, and had been found to have a non unctioning gallbladder. The diagnosis then vas biliary colic and obstructive jaundice, but ıer parents having refused permission for peration, the patient was discharged to her ıome. Pain radiated between her shoulders ınd was associated with chills and fever. She ıad had intermittent bouts of jaundice and ıoticed frequent passing of dark urine in the ix weeks before admission. Since her dis-harge in October 1959 the patient had been eeling rather weak and was not eating well.

Past history of this girl included an attack f pleurisy and pneumonia several years ago, ınd "ovulatory" difficulty in 1956. At that ime, she had been admitted to hospital with provisional diagnosis of acute appendicitis ıaving had a fever and some pain in the right ower quadrant. Her physical examination was hen normal and her leukocyte count was only 8600 of which 64% were neutrophiles and 25% band cells.

Physical examination at this time revealed a well developed girl presenting with epigastric ınd right upper quadrant tenderness and a positive Murphy's sign. Her Hb. level was normal and serum cholesterol was 145 mg. %. No spherocytosis was seen in the peripheral blood.

The patient was operated upon on January 12, 1960, and a small mucocœle of the gallbladder was found with no stones in the common bile duct. A cholecystectomy and an appendectomy were performed after stones were palpated in the gallbladder. Histological examination reported a subacute cholecystitis, cholelithiasis and chronic appendicitis. Her recovery was uneventful and convalescence at the time of writing has been uncomplicated.

CASE 3.—A 19 year old married woman was admitted to the emergency department of St. Paul's Hospital on March 7, 1960. She complained of severe upper abdominal crampy pain with nausea and vomiting. In the three months preceding admission she had suffered five similar attacks, unrelated to meals, bowel movements or micturition. Pain radiated to the mid-back in the right subscapular region. There was no history of dark urine or pale stools. The patient had had an appendectomy in the past and, except for an episode of jaundice in her youth, had always been well. She had no children. There was no familial history of blood disorders or splenectomies.

On admission, her temperature was 98.3°F. pulse 84 and blood pressure 118/70 mm. Hg. There was marked tenderness in the right subcostal area, and Murphy's sign was positive. Bowel sounds were present in all abdominal quadrants. A tentative diagnosis of acute cholecystitis was made.

A cholecystogram performed three weeks after admission showed no opaque stone, but the faintly concentrated dye was sufficient to outline several radiolucent calculi measuring about 5 mm. in diameter. The gallbladder contracted normally after a fatty meal. The results of routine laboratory tests on blood and urine were reported as being within normal limits. The test of erythrocyte fragility was normal. Serum bilirubin level was below 0.5 mg. %.

At operation on April 4, 1960, the gallbladder appeared moderately inflamed. It contained six mulberry calculi but none was found in the common duct. Pathological examination of the organ confirmed the clinical impression of chronic cholecystitis with cholelithiasis.

The postoperative course was smooth, although the apprehensive nature of the patient made mild sedation necessary.

DISCUSSION

A search of the records of St. Paul's Hospital, Vancouver, over the past 10 years discloses eight other legitimate cases pertinent to this article. A 16 year old girl was admitted with a diagnosis of cholecystitis and underwent cholecystectomy. The diagnosis was confirmed by microscopic examination of the gallbladder in which there were no stones. Seven other young women, between 15 and 20 years of age, had both cholecystitis and cholelithiasis. Of these, two had children and one of the patients was seven weeks postpartum at the time of operation. In these two patients, radiological examination had failed to reveal a functioning gallbladder. A stone was seen on the film of the 15 year old girl. Although she had been diagnosed as having hepatitis and appendicitis before admission, operation revealed cholecystitis with cholelithiasis and appendicitis. The gallbladder and appendix were removed and the patient made an uneventful recovery. A 20 year old woman who had hydrops caused by a stone in the cystic duct was cured by cholecystectomy with T-tube drainage of the common bile duct. The other three patients treated surgically were also women and all three had stones. In no case was familial

hæmolytic jaundice present in this series. There was no evidence of splenomegaly in any of the patients operated upon, nor was there any sign of hæmolytic anæmia in the reports of the laboratory tests performed before operation.

Five patients were treated conservatively. An 11 year old boy who had a non functioning gallbladder on radiological examination, responded to a fat free diet and other conservative therapy without any acute recurrence of his disease for at least 19 months (up to the time of writing). The only other patient suspected of having stones in the cystic duct was a two year old baby, whose radiological examination showed a non functioning gallbladder with calcified lymph nodes around the cystic duct. He was also treated conservatively and has not been readmitted in the past two years. The remaining three patients treated conservatively were all young women, two of whom were pregnant (a 17 year old mother at five months and a 19 year old mother at eight months). The third patient, who was four months postpartum, was icteric on admission. Pain, nausea and jaundice disappeared during her stay in hospital. Cholecystograms were performed on five of the conservatively treated patients after disappearance of their jaundice; in spite of double doses of contrast medium no gallbladder shadow could be detected.

Hospital records contained 20 additional cases of cholecystitis and cholelithiasis, but none of these was included in the present series, as they did not prove acceptable by our criteria. We included only cases in which disease was confirmed by pathological examination of the surgical specimen, or by failure of gallbladder visualization at radiography after administration of a double dose of dye, when jaundice had subsided.

Samson[19] reviewed cases of proven gallstone in 20 patients under 21 years of age operated upon at Hôtel-Dieu of Quebec; 18 of these were in girls or young women; the average age in this series was 16.3 years. Intercurrent infections were rare; one baby girl only three years old, appeared prone to develop influenza-like attacks. Another patient had a common bile duct exploration with Cattell tube in place for five months. None of these patients presented with splenomegaly or congenital hæmolytic anæmia.

Conclusion

The incidence of cholecystitis and cholelithiasis in the young may be higher than generally conceded; as more information is gathered on the various aspects of the malady, any dogmatism which may have been expressed in the approach to this problem in the past, is precluded.

It appears from the evidence presented above that gallstones are more common in young women than they are in young men under the age of 21. Cholelithiasis is more frequent in this section of the population than cholecystitis alone. In our operative series of 10 patients, nine had stones and only one had inflammation without calculus. None of our patients presented signs of hæmolytic anæmia. The gallbladder should be palpated and visualized at operation in all young patients suspected of suffering from appendicitis, regardless of mesenteric adenitis, or if the appendix looks normal. If this procedure were uniformly carried out, results might be startling.

The possibility of gallbladder disease should be kept in mind in all cases of this age group, presenting with abdominal ailments of obscure cause.

Summary

A short survey of cholecystitis and cholelithiasis in the preadult age group has been presented with mention of ætiological theories, symptoms, signs, routine investigation of diagnosis and therapeutic approaches. Three new cases are presented with a 10 year review from the files of St Paul's Hospital in Vancouver, and a similar 10 year survey from a hospital of approximately the same size in the Province of Quebec.

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Résumé

La littérature chirurgicale a rapporté jusqu'à maintenant, 500 cas de cholécystite, avec ou sans calcul, chez les jeunes, mais nous croyons son incidence plus élevée que ne le suggèrent ces rapports, à tel point que nous considérons la maladie dans le diagnostic différentiel des syndromes abdominaux obscurs de nos jeunes malades, même si n'est que pour l'exclure.

Du point de vue étiologique, il n'y a guère d'unanimité. L'anémie hémolytique congénitale, non plus que les maladies systémiques, doivent être considérées comme causales. Les pigments biliaires, plus que les sels, constituent les calculs.

Cliniquement, le jeune malade peut accuser un état nauséeux, des vomissements, de la douleur abdominale, de la diarrhée et de la jaunisse: celle-ci peut être présente sans calcul du cholédoque. La douleur irradiée est exceptionnelle.

Objectivement, l'examen peut être aussi souvent révélateur que négatif. On doit recourir à la cholécystographie au moindre doute. La cholécystectomie est le traitement de choix.

De la série des cas étudiés, il ressort que les calculs biliaires soient plus fréquents chez les jeunes femmes que chez les jeunes hommes de moins de 21 ans, et que la lithiase soit plus fréquente que la cholécystite simple; en effet, de nos 10 cas opérés, neuf avaient des calculs, alors qu'un seul présentait une inflammation sans calcul. Aucun de nos patients, en outre, ne manifestait d'anémie hémolytique.

HIATUS HERNIA AND PEPTIC ESOPHAGITIS FOLLOWING TRAUMA*

". . . Although a congenital short esophagus is mentioned frequently in surgical literature as a cause of hiatus hernia, in practice this is a very rare finding. The short esophagus usually is more apparent than real, easily reducible and is on the contrary *a posteriori* of hiatus hernia rather than its cause. The diagnosis of short esophagus is usually made following x-ray examination. The cardio-esophageal junction presents above the diaphragm and in association with a hiatus hernia of the sliding variety. This apparent short esophagus is probably due to the contraction of the longitudinal muscle fibers of the esophagus with a 'give' from the mobilized proximal end of the stomach. Although the radiologic diagnosis is a short esophagus, the surgeon has no difficulty in reducing the hernia and restoring the cardia below the hiatus unless esophageal stenosis and stricture resulting from peptic esophagitis is very far advanced.

"That the initial factors may be rather a weakness in the diaphragmatic crus, in the phrenoesophageal ligaments, in the so-called 'intrinsic esophageal sphincters', in the angulation of the esophagus into the cardia or in other structures whose integrity maintains the competence of the hiatus, or a combination of these factors, can only be postulated at the present time. . . ."

*Friedman, A. I.: *Am. J. Gastroenterol.*, **34:** 169, 1960.

CASE REPORTS

LEFT EPARTERIAL BRONCHUS: A RARE DEVELOPMENTAL ANOMALY OF THE BRONCHIAL TREE*

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ALTHOUGH a left eparterial bronchus is normally present in such animals as the camel, giraffe, hippopotamus and seal, such an arrangement of the bronchial tree of the left lung in man is rare. Brock[1] makes no mention at all of a left eparterial bronchus but states that the only common variant in the left upper lobe is "fusion of the pectoral and lingular bronchi". Boyden[2, 3] reported five cases of left eparterial bronchus and stated that to date he had not been able to find any previous reports in the literature. Wells[4] has added a further case report in which there was not only a left eparterial bronchus but a completely tri-lobed left lung, an exact replica anatomically of the right lung.

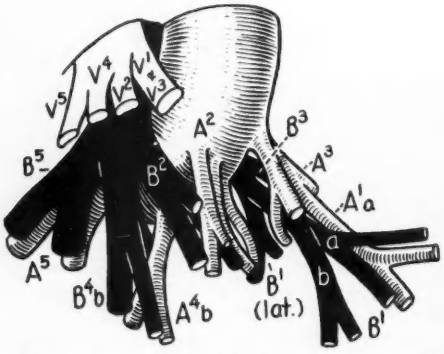


Fig. 1.—Reproduced from Boyden, E. A. and Hartman, J. F. (By kind permission of the publishers of the American Journal of Anatomy).

Boyden[2] believes that the anomaly is the result of the outgrowth of an ectopic bronchial bud high up on the left primary bronchus above the level of the developing left pulmonary artery. This ectopic outgrowth is followed by, or accompanied by the appearance of a lower division bronchus on the caudal side of the artery. The striking feature of the anomaly is the ectopic position of the left pulmonary artery which enters the left upper lobe between these two bronchial stems which arise separately from the left main stem bronchus (Fig. 1).

Boyden[2] described a case exactly similar to ours in which the left upper lobe was split by the pulmonary artery into two segments; the upper (or eparterial) stem supplied the apical and posterior segments and passed behind the left pulmonary artery; the lower stem passed anterior to the pulmonary artery to trifurcate into an anterior bronchus, and superior and inferior lingular bronchi (Fig. 2).

CASE REPORT

A 30 year old white man was admitted to

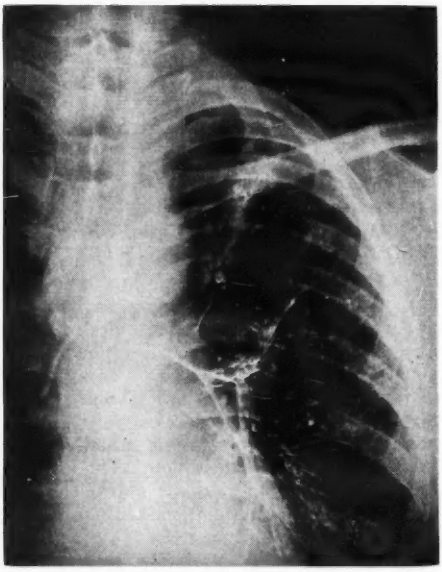


Fig. 2.—Bronchogram revealing eparterial bronchus supplying apico-posterior segment of left upper lobe.

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the Ongwanada Sanatorium, Kingston, with a one year history of recurrent hæmoptysis. Chest radiographs showed cavitation of the left apex. The sputum revealed tubercle bacilli by smear and culture. After five months of drug therapy cavitation persisted; therefore surgery was recommended. A preoperative bronchogram revealed a left eparterial bronchus supplying the cavitated apicoposterior segments (Fig. 2). Thoracotomy confirmed the arrangement mentioned above in that the left eparterial bronchus supplied the apical and posterior segments of the left upper lobe and this stem bronchus passed behind the left main pulmonary artery, while the stem bronchus supplying the remainder of the left upper lobe lay anterior to the artery. The diseased segment was removed in the usual manner and the postoperative course was uneventful.

Summary

A left eparterial bronchus is reported in a 30 year old man suffering from pulmonary tuberculosis. This rare anomaly was demonstrated by bronchography and confirmed by surgical resection. Although this is only the seventh case of left eparterial bronchus reported, it is probable that this does not represent the true incidence of the anomaly.

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Résumé

Un cas de bronche épartérielle gauche chez un homme de 30 ans qui souffrait de tuberculose pulmonaire est rapporté. Cette rare anomalie fut démontrée par bronchographie et confirmée à l'opération.

Même si ceci ne constitue que le septième cas de bronche épartérielle gauche rapporté, il est probable que cette anomalie ne soit pas représentée sous sa véritable incidence.

A REPORT OF 16 TUMORS OF THE SPINAL CORD IN CHILDREN: THE IMPORTANCE OF SPINAL RIGIDITY AS AN EARLY SIGN OF DISEASE*

"The occurrence of tumors involving the spinal cord in children is frequent enough to require the vigilance of all who are in pediatric practice. Commonly, early signs are missed and the diagnosis delayed until irreparable damage has been done. The apparent rarity of the disease is belied by the fact that more than a dozen articles related to it have been published during the last ten years.

"In most cases, the reward of diagnosis, even tardy, may be relief from intractable pain with complete remission of symptoms for many years or for life. Errors in diagnosis may result in progressive paraparesis and death.

"Although Ross and Bailey comment that 'the prime requisite for recognition of a spinal cord tumor is suspicion of its existence', it is rare for suspicion to be aroused without previous association with a case of spinal cord tumor. In the numerous reports in the literature the comment that tumors of the spinal cord may be about one-fifth as common as intracranial tumors under the age of 12 years would suggest that a pediatrician will rarely encounter such a tumor in his practicing life. If such a case does present, he may, in spite of careful examination and other investigations such as x-rays, fail to make the diagnosis and may even deny that the symptoms had any organic basis.

"The course of these tumors is too variable to permit an accurate estimate of prognosis either pre- or postoperatively, except on rare occasions. The major exception is when systemic disease, particularly of a malignant nature, has presented itself with involvement of the spinal cord or vertebrae. Thus the prognosis in any given child is individual and dependent upon the length of the history, the degree of neurological deficit, the nature of the lesion if determinable histologically, the extent of the tumor at the operation and the possibility of its removal, and the postoperative recovery and subsequent course. . . ."

*Richardson, F. L.: *J. Pediat.*, **57:** 42, 1960

PERICARDIAL FAT NECROSIS*

M. B. PERRIN, M.D., F.R.C.S.(Ed.), F.R.C.S.[C], *Winnipeg, Man.*

MEDIASTINAL TUMOURS and cysts are being diagnosed correctly more often than formerly because of improved radiological techniques and because thoracotomy is now a recognized diagnostic procedure. With experience, the type of mediastinal tumour or cyst can usually be diagnosed preoperatively from its anatomical location in the mediastinum. Thoracotomy may be necessary to make the final diagnosis. The following case of pericardial fat necrosis is reported because it represents a rare clinical entity and must be considered in the differential diagnosis of a mediastinal lesion.

CASE REPORT

A 56 year old man was referred to the Winnipeg Clinic on December 8, 1958, because a lesion had been discovered in the right chest on routine radiological examination (Fig. 1).



Fig. 1.—Routine radiograph showing lesion in right costophrenic region.

*From the Departments of Surgery of the Winnipeg Clinic, The Winnipeg General Hospital and the University of Manitoba, Winnipeg.

This patient had complained of a slight cough associated with a tickling sensation in the throat which had been present for a few years. He had no pain at the time of his first examination. However, two months earlier, he had an acute attack of pain in his right anterior chest below the right nipple. This pain radiated to the axilla and right supraspinatus area and lasted for 10 days. Deep breathing, sneezing and coughing aggravated the pain. He felt ill, but failed to take his temperature.

Physical examination was essentially negative. The laboratory findings were as follows: Urine—clear; hæmoglobin—15 g. %; sedimentation rate—40 mm. per hour. The radiological report on the chest was as follows: "There is an oval shaped density lying in the anterior inferior portion of the right middle lobe, measuring 8 x 5 x 5 cm. This does not seem to pulsate. The diaphragm moves freely in the region of the density, although its inferior margin cannot be separated from the diaphragm during fluoroscopy. However, it was seen that this mass changed shape as the diaphragm descended." A barium swallow showed that the œsophagus had no relationship to this mass and a barium enema failed to show large bowel herniation through the diaphragm.

The patient was admitted to the Winnipeg General Hospital on January 12, 1959, for bronchoscopy and further investigation of the mediastinal shadow. The tracheo-bronchial tree was found to be clear.

On January 15, 1959, a right thoracotomy was performed in the interspace between the fifth and sixth rib. On exploration the right lung was normal. There was a firm, yellowish mass lying in the diaphragm, adherent to the pericardium and to the base of the lower lobe of the lung. The mass was removed by freeing it from the base of the lung, the diaphragm and finally from the pericardium. Bleeding points were ligated, the lung inflated, and a rubber tube was placed in the pleural cavity. The chest wall was closed in layers.

The patient made an excellent recovery and was discharged from the hospital 10 days later. On his last visit to the Clinic in July 1959, he felt perfectly well and had no chest pain.

PATHOLOGICAL REPORT

"The tissue removed consisted of a well demarcated discoid mass, measuring 9 x 7 x

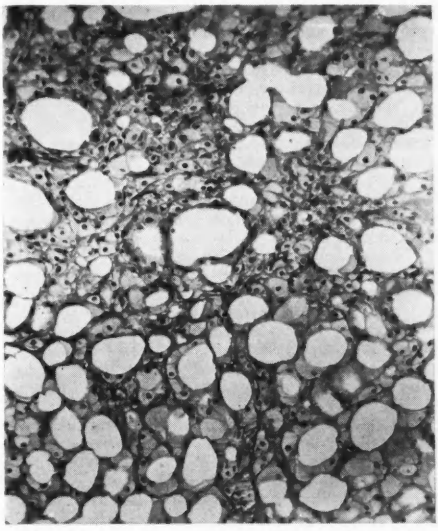


Fig. 2.—Low power photomicrograph to show general configuration of lesion (x 10).

1.5 cm. On section, it was variegated yellow and white. It appeared to be encapsulated and was firm to cut. Microscopically, it was limited by an ill defined capsule. The mass consisted of fat showing various stages of fat necrosis, but this process involved the whole lesion (Fig. 2). Of interest were numerous large vessels, mainly small arteries. Some of these showed intimal proliferation and others well marked medial hypertrophy (Fig. 3). The lumen in these vessels, though normal, seemed larger and out of proportion to the supplied area."

Discussion

This lesion has occurred in patients of middle age. The illness starts as an acute anterior chest pain of a pleuritic character and may even produce a pleural rub. After the acute phase the pain changes to a dull persistent ache. It has been reported[2] that pain was so severe that shock and a cardiac arrhythmia developed simulating a coronary occlusion. The radiographs of the chest demonstrate a mass in the cardiophrenic angle, spreading over the diaphragm, interpreted as lying in the area of the middle lobe.

Jackson, Clagget and McDonald[1] reported three cases and emphasized the vascular changes occurring in the pathological specimens of the surgically removed

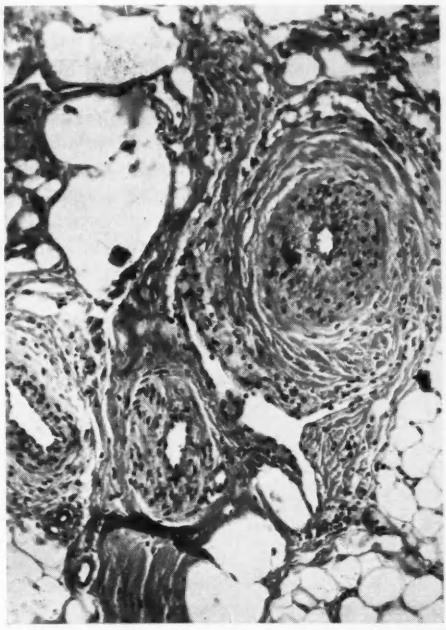


Fig. 3.—High power photomicrograph showing small arteries with intimal proliferation (x 90).

masses of fat necrosis. They considered various ætiological factors but concluded that they had no particular theory to offer.

Chester and Tully[2] described an earlier stage of the lesion, i.e. 13 days after the acute onset of symptoms. "Histologically, the changes were due to extravasation of blood, ischæmic fat necrosis and fibrosis."

Dr. Hugh Ross, pathologist at the Winnipeg General Hospital[3] made the following comments regarding this case: "There are two features of interest. The well defined capsule and the discrete appearance of the lesion is quite consistent with the diagnosis of a lipoma showing fat necrosis. The large abnormal vessels are suggestive of those found in hamartomata in various locations. It is interesting to speculate that these lesions begin as a tumour, such as a lipoma and because of the peculiar location, are subjected to the trauma of the beating heart and the moving diaphragm."

Summary

A case of fat necrosis involving the parietal pericardial fat is reported.

Pericardial fat necrosis is a new clinical entity that must be considered in the differential diagnosis of mediastinal masses.

The patient whose case is reported presented with a dull ache in the right anterior chest, following an episode of acute anterior chest pain eight weeks previously. The pain suggested a pleuritic lesion.

At thoracotomy, a mass of pericardial fat necrosis was found and removed.

To the best of our knowledge only four cases have been reported[1, 2] in the literature.

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Résumé

Avec l'expérience, le genre de tumeur médiastinale peut habituellement, selon sa localisation, être diagnostiqué pré-opératoirement quoique dans certains cas une thoracotomie soit nécessaire.

Un homme de 56 ans nous fut référé en décembre 1958 pour une lésion intra-thoracique droite découverte à la suite d'une radiographie de routine. Ce malade, qui n'accusait aucun malaise lors de l'examen, avait cependant éprouvé deux mois plus tôt, une violente douleur sous-mammelonnaire droite, irradiée à l'aisselle et à la région sus-épineuse droite, qui avait duré 10 jours et qui était accentuée par la respiration l'éternuement et la toux. Radiologiquement, il existait une densité ovalaire à la portion inférieure du lobe moyen droit qui n'était pas pulsatile, mais qui, à la fluoroscopie, changeait de forme avec les mouvements du diaphragme.

Une thoracotomie dans le 5e espace droit démontra un poumon normal et mit en évidence une masse jaunâtre ferme reposant sur le diaphragme et adhérente au péricarde et à la base du lobe inférieur. Cette masse fut enlevée par libération de ses adhérences.

Au microscope, cette masse possédait une capsule mal définie et était constituée de divers stages de nécrose graisseuse (Fig. 2). Elle montrait, de plus, de nombreux vaisseaux, surtout des petites artères, semblant hors de proportion avec le tissu à irriguer, ce qui amena le Dr Hugh Ross, pathologiste du "Winnipeg General Hospital", à commenter que ces vaisseaux anormaux rapellent ceux que l'on trouve dans les hamartomes.

Au meilleur de notre connaissance, seulement quatre cas de nécrose graisseuse péricardique ont déjà été rapportés.

JAMES HENRYSOUN: CHIRURGIAN TO THE POORE*

"James Henrysoun, barber surgeon, who practised in Edinburgh during the later sixteenth and early seventeenth centuries, has sometimes been referred to as the first Medical Officer of Health for the city. *Mutatis mutandis* —which is saying a good deal—there may be some justification for this, though in reality his position was rather that of a poor-law medical officer. Even during outbreaks of plague, when he was called upon for duties that today would be performed by the M.O.H., his functions were clinical rather than preventive. He belongs to a period when some, at least, of the larger communities were beginning to realize the advantage of having a medical man to whom they might apply for advice, even though the nature of his appointment and the extent of his duties might be somewhat loosely defined.

"Of Henrysoun as an individaul little can be said. He published nothing, he left no memoirs or correspondence, and his name is not associated with any special contribution to medical theory or practice. We know of him only that he made a reputation early in his career by his conduct during the great plague 1584-8, and that, thereafter, he seems to have stood high in the estimation of his professional brethren and of the magistrates of Edinburgh. On the other hand there is a fair amount of information about some of the matters with which he had to deal as an official. If he is, himself, a somewhat shadowy figure, the background against which he played his part is sufficiently clear to give a picture, not without interest, of the development of social medicine in Scotland in his time. . . ."

*Ritchie, J.: *Medical History*, **4**: 70, 1960.

CARCINOMA IN A THYROGLOSSAL REMNANT*

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DOUGLAS WAUGH, M.D., C.M., M.Sc., Ph.D., *Kingston, Ont.*

THE PURPOSE of this paper is to report what appears to be the 13th case of carcinoma arising in thyroglossal tract remnants. The literature on this rare lesion has been reviewed recently by Keeling and Ochsner[1] who found references to five cases and added two of their own. To these should be added the accounts of carcinoma arising in lingual thyroid recorded by Peracchia,[2] Tyler,[3] Marchal *et al.*,[4] Willis,[5] and Canciullo and Motta.[6] Other possible cases have been cited by Wapshaw,[7] but details on these are too scanty to allow evaluation of their exact nature. With so few recorded cases, it is not yet possible to discern any "standard" clinical and pathological picture, or to ascertain the biological behaviour of the lesion.

CASE REPORT

This 41 year old white woman was first seen in May 1959. At this time she complained of nervousness, hoarseness and dysphagia. In addition, she had noted a small lump in her neck in March 1959. On examination at this time, a small mobile mass could be palpated in the midline just below the thyroid cartilage. This moved with swallowing and with tongue protrusion. The lymph nodes in the neck were not enlarged. Laboratory studies revealed a serum cholesterol level of 249 mg. %, hæmoglobin 13.3 g. %, a blood leucocyte count of 6750/c. mm. with a normal differential count. Urinalysis demonstrated a specific gravity ranging from 1.010 to 1.025 with no abnormalities. A barium meal and chest radiograph were reported as negative. Bronchoscopy and œsophagoscopy failed to reveal any abnormalities in these areas.

On June 4, 1959, the cervical mass was exposed using a transverse skin crease incision, and the mass including the remnants of the thyroglossal tract were removed in toto. In addition, the pyramidal lobe of the thyroid gland was excised.

On frozen section at this time a tentative diagnosis of malignancy was made, and a careful search was made in the area for evidence of metastases. No enlarged nodes were found, and the thyroid itself was grossly free of tumour.

Pathological Findings

The excised tissue included the nodule, a portion of the pyramidal lobe and isthmus of the thyroid, and tissue from the thyroglossal tract. The nodule consisted of a bisected, irregular ovoid of firm, homogeneously yellow-white tissue on the external surface of which there were stringy fibrous tags. The halves of the nodule measured 1.2 cm. x 0.3 cm. x 0.5 cm. and 1.0 cm. x 0.3 cm. x 0.1 cm. respectively. Microscopic examination of this tissue revealed a relatively dense collagenous stroma, in one-third of which there were thyroid follicles containing colloid and lined by regular cuboidal epithelium. The remainder of the nodule consisted of small rounded or irregular aggregations of cuboidal or polyhedral cells closely resembling those of normal thyroid epithelium. In several of these alveolar

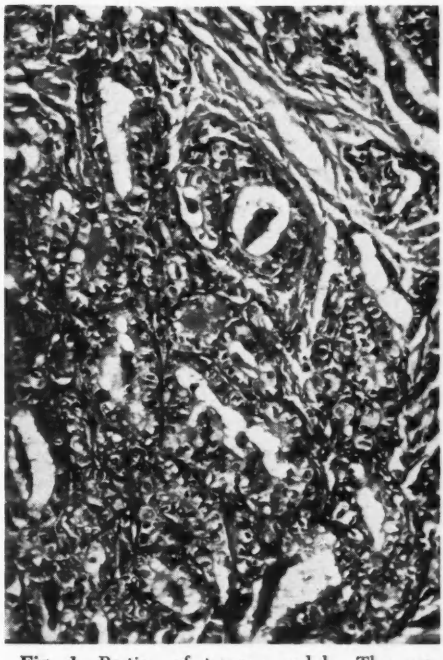


Fig. 1.—Portion of tumour nodule. The neoplastic follicles are irregular and lined by pleomorphic epithelium. Colloid occupies some of the lumina. Hemalum phloxin and saffron stain (x 123).

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clusters, there was a small central mass of eosinophilic colloid (Fig. 1). In sections stained by Verhoef's method for elastica, penetration of vessels by tumour tissue was convincingly demonstrated (Fig. 2).

Microscopic sections of the thyroid isthmus and pyramidal lobe showed normal thyroid structure and no evidence of tumour. Sections of the thyroglossal tract showed it to consist of a strand of connective tissue in which were isolated small nodules composed of thyroid follicles of regular morphology (Fig. 3). Three small lymph nodes were also present and were free of tumour.

The only neoplastic tissue found in the excised material consisted of the small fragment of well differentiated adenocarcinoma in the nodule that was clinically palpable. This was attached by a fibrous strand at one end to the thyroid isthmus and at the other end to the thyroglossal tract. The tumour was about 1.5 cm. above the apex of the pyramidal lobe of the thyroid.

Discussion

The diagnosis of carcinoma in a thyroglossal remnant in this case appears convincing. There was no detectable tumour in the thyroid proper, and in any event a midline suprathyroid route o metastasis would be most unusual. As with most neoplastic rarities, it is difficult to determine the factors of prognostic importance in this case. Up to the time of writing (eight months after operation) there has been no evidence of local recurrence, and it is our tentative conclusion that the prognosis here is probably the rather favourable one that is usually accorded well differentiated carcinomas arising in the thyroid itself. The rarity with which persistent thyroglossal remnants undergo neoplastic change suggests that while tumours may occasionally arise in heterotopic tissue this must be excessively rare in the case of this particular anomaly.

Of the six reported cases of carcinoma arising in lingual thyroid, it is noteworthy that five[2-6] were in men and one[8] was in a woman, all adults and of "middle age".[7]

Of the seven tumours arising in the thyroid tract proper, five were in women and two in men. Two of these tumours occurred in children.[1]



Fig. 2.—Tumour in the lumen of a vein. Outline of the vein wall is indicated by black elastica. An irregular mass of neoplastic epithelium fills the lumen. Verhoeff's elastic stain (x 123).



Fig. 3.—Nodule of normal thyroid in the connective tissue of the thyroglossal tract. Hemalum phloxin and saffron stain (x 47).

It is to be hoped that further examples of this rare condition will be reported and that from them more may be learned of the natural history of heterotopic thyroid carcinoma.

Summary

The 13th case of carcinoma arising in thyroglossal remnants has been described. The tumour consisted of a small midline suprathyroid nodule in a 41 year old woman. The good differentiation of the neoplasm and lack of extension other than by vascular invasion are suggestive of a favourable prognosis.

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Résumé

Cette étude a pour but de présenter le treizième cas de cancer reconnu à partir d'un vestige de canal thyréoglosse.

Il s'agit d'une femme de 41 ans qui, vue la première fois en mai 1959, se plaignait de nervosité, de voix rauque et de dysphagie et qui présentait une petite tuméfaction cervicale depuis mars 1959. Objectivement, on pouvait palper sous le cartilage thyroïde une petite masse mobile qui était entraînée avec la déglutition et la protusion de la langue. Tout autre examen complémentaire se révéla négatif.

L'opération consista en l'exérèse complète de la masse avec inclusion du lobe pyramidal. Une coupe par congélation per-opératoire étant suggestive de malignité, déclencha un examen minutieux régional qui s'avéra négatif tant du côté ganglionnaire que thyroïdien.

A l'examen macroscopique, le nodule qui avait un aspect ovoïde irrégulier, montrait une surface de section blanc-jaunâtre homogène traversée de cordes fibreuses.

Au microscope, la coupe montrait du collagène relativement dense avec des follicules thyroïdiens à contenu colloïde et tapissés d'un épithélium cuboïde d'une part, alors que le reste montrait des aggrégations irrégulières ou arrondies de cellules cubiques ou poly-hédriques ressemblant étroitement à un épithélium thyroïdien normal d'autre part.

Plusieurs de ces amas alvéolaires montraient une masse centrale de colloïde éosinophile (Fig. 1). Sur des coupes préparées par la méthode de Verhoef pour montrer la limitante élastique interne, l'envahissement des vaisseaux était clairement démontré (Fig. 2).

Les coupes du lobe pyramidal et de l'isthme thyroïdien, celles du canal thyréoglosse, de même que celles de trois ganglions lymphatiques étaient sans particularité.

Le seul tissu néoplasique, en l'occurence un épithélioma folliculaire bien différencié, était contenu dans le nodule cliniquement palpable qui était localisé entre l'isthme thyroïdien et le canal thyréoglosse.

Des sept cas publies par Keeling et Ochsner, il semble que cette lésion favorise la femme dans la quarantaine et ce cas-ci diffère seulement dans sa classification pathologique puisque cinq des cas publiés étaient des épithélioma papillaires.

PROSTHETIC PRINCIPLES – ABOVE KNEE AMPUTATIONS. Miles H. Anderson, Director, Prosthetics Education Project, School of Medicine, University of California; John J. Bray, Associate Research Prosthetist, School of Medicine, University of California, and Charles A. Hennessy, Associate Director, Prosthetics Education Project, School of Medicine, University of California. 331 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $11.00.

This is a worthwhile book of practical value for prosthetists and limb-fitting surgeons. The background knowledge on which the volume is based is from the "Prosthetics Education Project" of the University of California. This project has accomplished a great deal in furthering the knowledge of gait and mechanics in above knee amputees. The information obtained has been recorded in accurate detail, and records step by step procedure in fitting and making the various above knee prostheses.

It is unlikely this volume would hold much interest for most surgeons, but it would be well worthwhile for all prosthetists and the occasional surgeon dealing with large numbers of amputees.

MYCOTIC ARTERIAL ANEURYSMS

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THE DIAGNOSIS and treatment of arterial aneurysms has received recent prominence in the surgical literature due to the marked advances in management. Arteriosclerotic aneurysms comprise the great majority, traumatic types appear to be increasing, while the syphilitic varieties are becoming extremely rare in this country. Mycotic arterial aneurysms are seldom diagnosed before complications occur and are rarely considered as a type of aneurysmal disease. This point is understandable as this variety is uncommon, as illustrated in our experience at the Royal Victoria Hospital, Montreal where, to date, we have repaired or excised and grafted 58 aneurysms of all types. Fifty-six were arteriosclerotic, one probably syphilitic and one mycotic. A discussion of this case and a review of the associated literature on this subject forms the basis of this report.

ÆTIOLOGY

The infective basis of mycotic aneurysms was first recognized by Goodhart[12] in 1877 when he pointed out the relationship of these aneurysms to endocarditis. He states "the clot detached from such a focus will poison the part in which it is lodged and lead to acute softening of the arterial wall by inoculating it with its own inflammatory nature." In 1887 Eppinger[7] was able to demonstrate the same bacterial organisms in the wall of the aneurysm as were present in the cardiac valvular vegetations. This has been subsequently corroborated by others,[3, 19] although in most instances it is difficult to obtain a positive culture from the arterial wall.

Infectious origins other than subacute bacterial endocarditis are rare though reports of foci in bones and lung appeared in the pre-antibiotic era. For instance, in 1923 Stengel and Wolferth[27] reported 187 out of their 217 collected cases of mycotic aneurysms as occuring in association with endocardial disease, the remaining 30 with a variety of other infections. Rheumatic fever is the most common ætiological factor in the development of the endocarditis and in the series of Shnider and Cotsonas,[2] of 42 patients with mycotic aneurysms, 36 had endocarditis subsequent to rheumatic heart disease.

The organism most frequently recovered from the blood stream, the heart valves or the aneurysm is the streptococcus. Table I shows in descending order the relative frequencies of the different bacteria involved.

PATHOGENESIS

Infection of the arterial wall takes place by one or more of the following mechanisms: (1) Direct spread of bacteria from adjacent septic lesions. (2) Lodgement of infected emboli in the lumen of the artery (Eppinger[7]), or the vasa vasorum (de Takats[6]). (3) Settling of the bacteria on the intimal surface. (4) Continuity or contiguity of infection from the cardiac valves.

The acute or subacute arteritis that follows invasion by bacteria results in swelling, ischæmia, splitting and fragmentation of the elastic tissue. Weakening with dilatation of the arterial wall is then inevitable. Once the initial dilatation has taken place the course is steadily progressive. Violent physical exertion or emotional stress are frequently factors precipitating rupture of the aneurysm.[14]

The "incubation period" of mycotic aneurysms will depend on the severity of the infection, virulence of the organism, host resistance and treatment. The location of the reported mycotic aneurysms is shown in Table II.

Since subacute bacterial endocarditis involves the left heart in the great majority of cases,[3, 12, 26] it follows that mycotic aneurysms occur, for the most part, in the systemic arterial tree. The pulmonary arterial circulation may become the site in those infrequent cases of right sided endocarditis or when there is an abnormal communication between the two circulations

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TABLE I.—Bacterial Organisms Associated with Mycotic Aneurysms

Stengel and Wolferth[27]*—1923*	*Shnider and Cotsonas*[26]*—1954*	*Anderson*[1]*—1957*
Streptococcus (chiefly non hæmolytic)	Streptococcus viridans	Streptococcus
Staphylococcus	Streptococcus salvarius	Pneumococcus
Pneumococcus	Streptococcus hæmolyticus	Gonococcus
Influenza bacillus	Staphylococcus	Influenza bacillus
Gonococcus		Typhoid bacillus
		Others

is in the case of a patent ductus arteriosus. Arterial emboli from the valve vegetations may be of sufficient size to occlude relatively major arteries and occur frequently in association with mycotic aneurysms.

Most aneurysms are small, varying in size from that of a millet seed to that of a pea. Vessels of all sizes are involved from the smallest unnamed arteries to that of the aorta. Only occasionally will the aneurysm size exceed a diameter of 2 cm. to 3 cm. with the exception of the false aneurysm produced by rupture. Vegetations similar to those of the heart valves may be present about the edges of a mycotic aneurysm or in its depth.

Histologically there is loss of intima and destruction of elastic tissue, including the internal elastic lamina, acute or subacute periarteritis and mesarteritis. Polymorphonuclear leucocytes are abundant with masses of bacteria. In the more acute aneurysms, there is marked infiltration with polymorphonuclear cells and microscopic abscesses are not uncommon.

TABLE II.—Distribution and Incidence of Vessel Involvement in Mycotic Aneurysms

	Stengel and Wolferth	*Shnider and Cotsonas*
Aorta	66	5*
Innominate	2	
Vertebral	1	
Basilar	4	3
External carotid		1
Internal carotid	3	
Cerebral	34 (middle cerebral 14)	13 (middle cerebral 3)
Subclavian	1	
Axillary	3	1
Brachial	10	1
Radial	5	
Ulnar	5	3
Iliac	10	5
Gluteal	3	3
Femoral	16	6
Profunda femoris	2	
Popliteal	5	4
Posterial tibial	8	5
Coronary	9	
Superior mesenteric and branches	24	14
Splenic and branches	15	
Hepatic and branches	19	2
Renal and branches	5	
Pulmonary and branches	14	3
	264	69

*Excludes proximal aortic arch.

Clinical Considerations

A study of the reported cases and our own experience reveals that mycotic aneurysms have certain rather characteristic features which aid in distinguishing them from other varieties.

1. They occur in the course of a bacteræmia, usually associated with bacterial endocarditis.

2. They are frequently multiple. Approximately one out of every four cases reported by Stengel and Wolferth[27] had more than one aneurysm.

3. The peak age incidence is in the third decade. In a report of 62 cases by Shnider and Cotsonas,[26] the youngest patient was eight years and the oldest 50 years of age.

4. Mycotic aneurysms are commoner in men in a ratio of 3:1, despite the fact that rheumatic mitral disease is twice as common in women.[24]

5. Pain precedes their appearance, being intense and at times difficult to localize.

6. Rupture occurs frequently before the aneurysm reaches any appreciable size. In an extremity this produces a false sac usually with self-limited bleeding in contradistinction to the free exsanguinating hæmorrhages of intraabdominal and intrathoracic types.

7. These aneurysms commonly occur at bifurcations.

8. The proximal aorta is a favourite site for mycotic aneurysms, but the abdominal aorta is infrequently involved. Only seven cases could be found in the literature of abdominal aortic involvement.[20, 29] The superior mesenteric artery is much more frequently involved than any other major

branch artery. One such aneurysm has been resected by DeBakey and Cooley[5] but the majority are found at autopsy because of diagnostic difficulties.

Shnider and Cotsonas[26] have described four clinical syndromes by which ruptured mycotic aneurysms may reveal themselves.

1. Intracranial syndrome: These patients develop a subarachnoid hæmorrhage, hemiparesis or hemiplegia and coma. Increasing headache is commonly a preceding symptom.

2. Abdominal syndrome: Abdominal pain and collapse with a shock-like state and signs of intraabdominal or extraperitoneal hæmorrhage in a patient with or following subacute bacterial endocarditis, should indicate this possibility. A pulsating mass with a bruit rarely occurs.

3. Thoracic syndrome: Severe chest pain, hæmoptysis and shock indicates the possibility of rupture. Hæmothorax may be present but relatively sudden death is the rule.

4. Extremity syndrome: Rupture of an aneurysm in an extremity is usually contained by the surrounding tissues with the development of a false sac. The sudden development of a pulsating mass is strongly suggestive of this lesion. An associated bruit is continuously heard. The rupture is accompanied usually by severe continuous pain due to local tension and nerve compression.

As a mycotic aneurysm projects from one aspect of the wall, the flow in the artery is seldom interfered with unless the pressure of the false sac is such as to occlude the distal flow. Therefore, thrombosis in the false sac rarely involves the parent artery with the result that gangrene subsequent to thrombosis or rupture of this type of aneurysm is much less common than it is in the arteriosclerotic variety where the aneurysm is in the form of a fusiform involvement of the affected artery.

Radiography and arteriography are valuable diagnostic aids. Large densities in the chest may be visualized on plain films but arteriography is necessary to accurately diagnose the intracranial or abdominal types. Clinical examination is usually sufficient for those in the limbs but arteriography adds further detail regarding location and size.

Prognosis

Due to the difficulty or virtual impossibility of diagnosing a mycotic aneurysm prior to rupture the prognosis is grave. In the intracranial types a lesser initial leak may allow the diagnosis to be made by cranial arteriography with a good chance of subsequent surgical cure. As mentioned above, rupture of the extremity type is readily diagnosed and can be treated successfully if found sufficiently early.

Mycotic aneurysms have been reported[27] to undergo spontaneous cure by thrombosis of the aneurysm or the false aneurysm produced by rupture. The incidence of such "cures" is impossible to estimate but may be commoner than is believed because the number of cases of subacute bacterial endocarditis developing clinical evidence of an aneurysm is quite low.

Treatment

Before the use of antibiotics, endocarditis had an extremely high mortality and a mycotic aneurysm only added to the poor outcome. As the prognosis of the underlying disease has improved with antibiotic therapy coincident with the improvement in vascular surgical techniques, the mortality rate in the intracranial types and particularly in the extremity group has shown a marked improvement.

In all aneurysms, excision and restitution of normal blood flow by grafting is the ideal objective. However, inasmuch as the mycotic aneurysm is a lateral variety (even when ruptured with a false sac), the surgical problem is somewhat different from that of the usual arteriosclerotic type. The need for resection and grafting of any appreciable length of the parent artery rarely arises because the originating artery seldom shows complete occlusion.

The main vessel proximal to the aneurysm should initially be cleared and controlled by a clamp. The false sac is opened as dissection of this sac is not possible due to the fact that it is composed only of thickened surrounding tissues. Through the opened sac the mouth of the aneurysm in the host artery can be closed by

TABLE III.—Reported Cases of Mycotic Aneurysms Treated Successfully by Operation

Year	Author	Site	Underlying illness	Procedure	Result
1908	Clutton and Dudgeon[4]	Femoral	Pneumococcal pneumonia	Proximal ligation	Satisfactory
1909	Lewis and Schrager[18]	Brachial	Subacute bacterial endocarditis	Proximal ligation	"
1934	Gage[9]	Common iliac	"	Sympathectomy proximal ligation	"
1946	Klein and Crowell[17]	Ulnar	"	Excision	"
1948	Hurwitz and Arst[15]	Brachial	"	Excision	"
1948	Julian[16]	Common femoral	"	Excision	"
1949	Goadby *et al*[11]	Femoral	"	Proximal ligation	"
1951	Rogers[25]	Femoral	"	Sympathectomy ligation and division of external iliacs: aneurysmorrhaphy	Good
1952	Fernbach *et al*[8]	Common iliac	"	Ligation	"
1953	DeBakey and Cooley[5]	Superior mesenteric	"	Resection: simultaneous ligation of mesenteric vein	"
1953	Harrison and Desmond[13]	Popliteal	"	Proximal ligation	"
1953	Tresidder and Warren[28]	Posterior tibial (Bilateral)	"	Excision	"
1954	Barker[2]	Deep femoral (Bilateral)	"	Excision	"
1955	Moore and Telling[21]	Common iliac	"	Resection of aortic bifurcation and graft with preserved arterial graft	"
1956	Rogers[23]	Femoral (bilateral)	"	Hunterian ligation and division of external iliacs	"
1957	Ghabrial and Higazi[10]	Common femoral (bifurcation)	"	Sympathectomy: excision and arteriorrhaphy	"
1960	Siwak and Luke	Popliteal	"	Proximal ligation and obliterative aneurysmorrhaphy	"

interrupted silk sutures. If this procedure is only partly successful in controlling the aneurysm a proximal ligation of the associated main vessel is performed. As in most aneurysms, a large layer of clot lines the false sac and this clot may have extended into the aneurysmal ostium to occlude the parent artery. Usually this clot extension will readily lift out with the aneurysmal layers, but occasionally distal propagating thrombosis will require removal by wire loop or flushing through an opening in the artery distal to the lesion.

Lumbar sympathectomy will occasionally tide over a dubiously viable extremity where proper restoration of blood flow has not been achieved.

Table III summarizes the location, types of procedure and results in the reported series of mycotic aneurysms successfully treated to date.

Case Report

C.B. a 59 year old labourer, was admitted to a neighbouring hospital on October 27, 1959, with a two week history of chills, fever and neck pain. Examination revealed no explanation of his febrile illness and his spinal fluid examination was negative. On November 18 he developed pain in the left knee and the left foot became swollen, red and painful. Subacute bacterial endocarditis was considered but blood culture was negative and his heart sounds were normal.

On November 27, an aortic diastolic murmur was heard for the first time while his diastolic pressure dropped from 80 on admission to 45 mm. Hg. In addition, there were bouts of extrasystoles. He was started on 5,000,000 units of penicillin daily and his fever gradually disappeared. The heart murmur however, became more prominent. Tenderness in the left popliteal fossa developed

along with fulness in the upper calf. The foot pulses were strong and equal.

A blood culture done on December 1, was positive for *strep. viridans* and the penicillin dosage was increased to 10,000,000 units per day. During the latter part of December, pulsations at the left ankle became weaker while pain in the left calf increased to a degree where opiates were necessary. A pulsatile mass appeared in the upper calf and popliteal region which revealed a large dumbbell shaped aneurysm on arteriography. The patient was transferred to the Royal Victoria Hospital on January 8, 1960.

Examination revealed an irregular pulse of 84 per minute, B.P. 110/50 and normal temperature. Cardiac examination revealed an early, high pitched diastolic murmur over the entire precordium, best heard to the left of the sternum. A faint rumbling middiastolic murmur was present confined to the cardiac apex. The left leg showed a large round pulsatile mass about the size of an orange underlying the superior portion of the gastrocnemius muscle and accompanied by a loud bruit. The left dorsalis pedis pulse was faintly palpable and the posterior tibial pulse was absent.

Laboratory examinations revealed a normal urine, Hb. 10 g. %, N.P.N. 28 mg. % and cholesterol 123 mg. %. A blood culture was negative. Further arteriography (Fig. 1) disclosed the large aneurysm and a diagnosis of ruptured mycotic aneurysm with false sac was made.

Operation was performed on January 12, 1960. The thin sac wall was densely adherent to the adjacent muscle and could not be separated. The aneurysm was opened after control of the proximal popliteal artery and retrograde bleeding occurred from an opening deep in the sac which was presumed to be the point of rupture of the previous mycotic aneurysm. This opening was about 1 cm. in length and was closed by interrupted sutures from the aneurysmal side. To be certain of hæmostasis, a ligation of the popliteal artery just proximal to the aneurysm was performed. On opening the aneurysmal sac a large cast of thrombus filling the sac was evacuated. Because the sac wall was impossible to remove, an extensive dead space remained which extended from the flexor compartment



Fig. 1.—Arteriogram revealing the large dumbbell shaped sac of the false aneurysm.



Fig. 2—Ten day postoperative healed incision showing the degree of exposure necessary.

through the interosseus ligament to the anterior space. This was filled by a pedicled muscle flap taken from the soleus muscle and the wound was drained. Following operation, the left foot was cool and dusky but within three days regained normal colour and a faint dorsalis pedis pulse could again be felt. All the patient's previous leg pain had disappeared.

Microscopic report of a section of the wall of the aneurysm reads as follows "the wall of the aneurysm shows hyaline degeneration. The adventitial tissue is thickened by dense fibrosis and focal and chronic inflammatory cell infiltrate. Some fibrosed muscle is attached. The thrombus has layers of old and more recent thrombosis." Further blood cultures on January 22 and 26 were negative. The healed operative incision is shown in Fig. 2.

Since discharge from hospital, the patient has been free from pain. He has claudication in the left foot on walking more than four blocks at a fast pace, but reports progressive improvement in this symptom.

Summary

A case of mycotic aneurysm of the popliteal artery complicating subacute bacterial endocarditis is presented in which successful proximal ligation and modified obliterative endoaneurysmorrhaphy was carried out.

The ætiology, pathogenesis, incidence, diagnosis and treatment are reviewed in the light of our experience and that of the world literature.

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RÉSUMÉ

Les anévrysmes mycotiques artériels sont exceptionnellement diagnostiqués avant que surviennent les complications, et à cause de leur incidence sont rarement inclus dans la maladi anévrysmale, ce qui est illustré par notre ex périence au "Royal Victoria Hospital" où jusqu' maintenant nous avons réparé ou excisé et greff 58 anévrysmes, dont 56 étaient artériosclérotique un probablement syphilitique et un mycotique.

L'origine infectieuse des anévrysmes mycotique fut premièrement reconnue par Goodhart[12] e 1877 lorsqu'il fit la relation entre les anévrysme et l'endocardite. L'organisme le plus souvent e cause est le streptocoque.

Cliniquement, ils surviennent au cours d'un endocardite bactérienne sub-aiguë et sont souven multiples. Ils donneront, selon leur localisatio un syndrome douloureux prémonitoire du dram

Le pronostic est toujours grave, mais depui l'avènement des antibiotiques et l'avancement d la chirurgie vasculaire, le taux de mortalité a diminué en ce qui concerne les localisations intracrânienne et en particulier aux membres.

L'excision associée à la greffe artérielle pour rétablir le flot sanguin donne les meilleurs résultats.

Un cas d'anévrysme mycotique de l'artère poplitée survenu au cours d'une endocardite bactérienne sub-aiguë avec correction chirurgicale est rapporté.

JENNER AND VACCINATION*

"Edward Jenner's great contribution to vaccination against smallpox stands out, perhaps unequalled in the domain of public health and preventive medicine. But both Jenner's work and Jenner's personality were variously judged at the time. It was claimed that vaccination was based on unsound grounds and that Jenner himself was little more than a quack. The idea that an attack of cowpox safegarded the victim against smallpox was said to be prevalent among the dairy hands in Gloucestershire, and Jenner was a Gloucestershire boy. In 1796 Jenner inoculated the skin of a lad, James Phipps, with material from a cowpox vesicle on the hand of a dairymaid, Sarah Nelmes. The vaccination took, and later inoculation of smallpox material failed to produce disease. . . .

"Perhaps it is fairest in appraising the rationale of Jenner's work to stick to his published facts as exposed in the 'Inquiry'. After an introductory paragraph, Jenner turns immediately to discussion of a disease of horses called 'the grease'. 'It is an inflammation and swelling in the heel, from which issues matter possessing properties of a very peculiar kind, which seems capable of generating a disease in the Human Body (after it has undergone the modification which I shall presently speak of) which bears so strong a resemblance to the Small Pox, that I think it highly probable it may be the source of that disease.' Jenner goes on to claim, in brief, that milkers may contaminate cows with material from 'the grease', which produces cowpox, which, in turn, may be transmitted to the milkmaids. 'Thus the disease makes its progress from the Horse to the nipple of the Cow, and from the Cow to the Human Subject . . . but what renders the Cow-Pox virus so extremely singular is, that the person who has been thus affected is forever after secure from the infection of the Small-Pox; neither exposure to the variolous effluvia, nor the insertion of the matter into the skin, producing this distemper.' . . .

"The great fact stands out that Jenner's thesis has turned out to be correct, but was this a lucky guess, as some have claimed, or does his published evidence really justify his conclusions?" . . .

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*BLOOMFIELD, A. L.: Some footnotes to Medical History, *A.M.A. Arch. Int. Med.*, **106:** 293, 1960.

SIGMOIDOSCOPIC APPEARANCE OF COMMON ILIAC ARTERY ANEURYSM

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During sigmoidoscopic examination it is not uncommon to see or feel the pulsations of the iliac arteries, but during a recent examination an iliac aneurysm was thus diagnosed. Consultation with colleagues and a search of the literature have failed to reveal any record of a similar finding.

Case Report

W.S., a white man aged 71, was admitted to hospital on October 28, 1959. He complained of colicky central abdominal pain of four days' duration associated with absolute constipation. For three days he had not passed flatus. He had been belching, but had not vomited. He was habitually somewhat constipated. There was no blood in his stools or melæna. He was known to be hypertensive, with his highest recorded blood pressure 222/154.

On examination the patient was obese; his blood pressure was 166/88; his temperature and pulse were normal. The abdomen was somewhat distended, with normal bowel sounds, and an ill defined tender mass below the left costal margin. No other masses or pulsations could be felt in the abdomen. The femoral artery pulsations were normal. Digital rectal examination was negative.

On October 29, 1959, sigmoidoscopic examination was performed with the patient first in the left lateral and then the knee-chest position. *A large pulsatile swelling was seen and felt through the bowel wall on the right side at six inches*, which suggested the diagnosis of an aneurysm of the right iliac artery and warranted caution in the examination. Spasm of the rectosigmoid junction prevented examination beyond seven inches, but no other findings were noted.

On October 30, 1959, a flat plate of the abdomen showed no abnormalities, and a barium enema revealed diverticulosis of the sigmoid colon. Hæmoglobin was 13.1 g. % but on November 1, 1959, the white cell count was 20,600 per cu. mm.

At 10.30 p.m. on November 1, 1959, the patient complained of sudden excruciating pain in the left lower quadrant of the abdomen, and promptly became extremely shocked, pulseless and perspired profusely. In spite of resuscitative measures he died 40 minutes later.

At postmortem examination there was an extensive left sided retroperitoneal hæmatoma and about 500 c.c. of free bloodstained fluid in the peritoneal cavity. The abdominal aorta from the renal arteries downwards, was hard and dilated to 10 cm. diameter. On the left postero-lateral wall of this aortic aneurysm there was a penetrating tear 3 cm. long. Both common iliac arteries were dilated to 4 cm. diameter to a point 8 cm. distal to the aortic bifurcation (Fig. 1). There was an old mural thrombus of the aortic wall, and a recanalized central thrombus of both common iliac arteries. The descending branch of the left coronary artery was half occluded by an atheromatous plaque. There were numerous diverticula of the sigmoid colon.

Discussion

In view of the patient's obesity, the

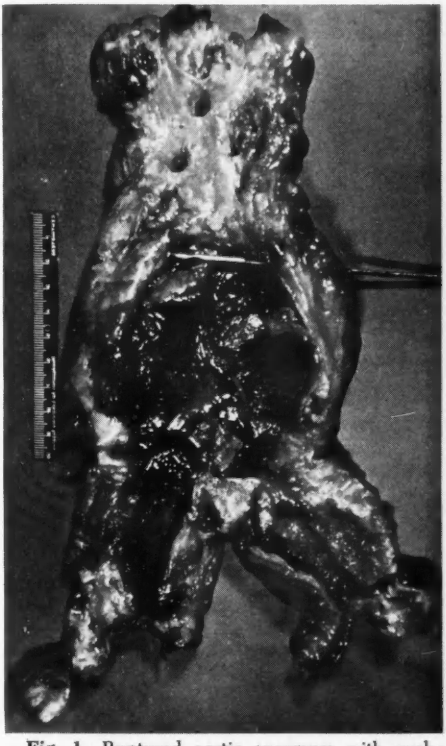


Fig. 1.—Ruptured aortic aneurysm with probe through ruptured portion on left side and aneurysms of both common iliac arteries.

abdominal aneurysm could not be felt through the anterior abdominal wall. The diagnosis of iliac aneurysm was made as a result of the sigmoidoscopic examination. Prompt recognition of such an aneurysm is important to prevent rupture of the aneurysm during the examination.

SUMMARY

A case is described in which a right common iliac aneurysm was diagnosed at sigmoidoscopic examination and later confirmed by postmortem examination, when a ruptured abdominal aortic aneurysm was found.

ACKNOWLEDGMENTS

We wish to thank Dr. W. W. Ridge fo referring this case; the pathology department o St. Joseph's Hospital, Hamilton, for the post mortem report and photograph; and Miss J Ashley for preparation of the colour photograp for monochrome reproduction.

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RÉSUMÉ

Un cas est rapporté donc l'examen sigmoido scopique a présenté un aneurisme de l'artèr moyenne iliaque. On ne peut pas palper l'aneu risme dans l'examen de l'abdomen parce que l malade était trop gros. Le diagnose était confirm après trois jours quand le malade est mourit cause d'un rupture d'un aneurisme aortiqu abdominal.

ARTIFICERS OF THE GOLDEN BOWL*

". . . By the time of (Horsley's) death, Harvey Cushing in America had established himself as the pioneer of neurosurgery in that country. His genius and his contribution were of a different kind, and in historical orientation he will probably be regarded as the cartographer of this new world into which Horsley had sailed, and as the one who brought its treasures from the prerogative of genius and placed them in the safe keeping of lesser men.

"A graduate of Yale, Cushing had received his first stimulus from Jack Elliott, of the Massachusetts General Hospital, when he was an intern. In 1895, Jack Elliott, acting under the stimulus of Horsley, essayed the first operations on the brain in the United States of America. These had proved rapidly fatal. Cushing had been very interested in these cases, and characteristically had made notes on them. Elliott suggested to him that they should be reported; but Cushing refused to cooperate, feeling that this dismal story would be poor publicity for a branch of surgery to which he had already felt the call.

"From earliest years, Cushing had chosen paths leading to success. An early comment made by Professor Newell, at one time a fellow resident, hints at his consecration to success:

'He was recognized as the ablest man of his class, and an extremely hard worker. As a house officer, I was his junior and suffered severely in that position for a year. He was an extremely hard man to work with, whether one was over him or under him, as his tremendous ambition for success made it impossible for him to allow anyone else to get credit for work done. As you know when he wanted to be, he was one of the most charming people in the world, but working with him I found he couldn't tolerate anyone else in the limelight.' . . ."

*MILLER, D.: *M. J. Australia*, **2**: 5, 1960.

NEONATAL RUPTURE OF THE STOMACH DUE TO CONGENITAL MUSCLE DEFECT*

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Introduction

Perforation of the stomach in the neonatal period is rare. Publications dealing with this condition have appeared infrequently in recent years and these have indicated that too often the condition is not recognized until postmortem studies have been made. Failure to consider the possible existence of this disorder often leads to delay in diagnosis which has contributed markedly to the very high mortality. If these unfortunate babies are to have any chance of survival, early diagnosis and early operation are mandatory.

Siebold[14] was the first to report on gastric perforation in a newborn infant in 1825. Since then over 70 cases have been reported in the literature. The first attempt at surgical repair of a perforation of the stomach in a newborn infant was described by Stern[17] in 1929. The first newborn infant to survive operation was reported by Legar[8] in 1950, although in 1932 Selinger[13] had successfully repaired a gastric perforation in a three month old baby.

Only one case of spontaneous gastric perforation in a newborn infant is on record at The Montreal Children's Hospital. Features of the case are so typical of the majority of those in the literature that its presentation seems worthwhile.

Case Report

The patient was born on August 26, 1958. The mother had had one previous normal pregnancy. On this occasion she was delivered one week before term, the delivery being spontaneous and uneventful.

The baby was an apparently normal boy weighing 7 lb. 3 oz. (3260 g.). He took his early feedings well and meconium was passed spontaneously. On August 29, his temperature was 100.3° F. but dropped to 98.4° F. in the evening, and on August 30 it remained at 99.2° F. throughout the day. That same evening after a feeding, he vomited some bile stained fluid. He appeared pale and slightly cyanotic and his respiratory rate was rapid. There was no abdominal distension and the bowel sounds were normal. He took other feedings normally during the night.

On the morning of August 31, his fifth day of life, considerable deterioration in his condition was observed. Along with respiratory distress he now had marked abdominal distension and no bowel sounds were audible. At this stage he was transferred to The Montreal Children's Hopsital.

On admission, physical examination revealed a lethargic infant with mottled cyanosis, most pronounced in the extremities, and severe abdominal distension. The temperature was 100.6° F. and the pulse rate was 160 per minute and regular. Although the respirations were rapid and irregular, the chest was clear to auscultation. The abdomen was tympanitic. No masses were palpable although the baby resented abdominal palpation.

Radiological examination demonstrated no abnormality in the chest, but revealed a considerable amount of free air in the peritoneal cavity and very little air in the gastrointestinal tract (Figs. 1a and 1b).

Continuous nasogastric suction and intravenous infusions were started. Antibiotics and vitamin K were given. The infant was taken to the operating room two hours after admission to hospital.

At operation, air escaped as the peritoneal cavity was entered. A large quantity of yellow bile stained fluid was aspirated. On general inspection no gross lesion was visible. More searching examination indicated that the fluid was oozing from the foramen of Winslow. The gastrocolic omentum was divided, the lesser sac entered, and a perforation was found high on the posterior wall of the stomach towards its greater curvature. This opening appeared to be 4 cm. to 5 cm. in length. It was closed by means of a single layer of interrupted and inverted mattress sutures of fine black silk. The abdominal incision was closed in layers and a small Penrose drain was left in the lesser sac.

The baby's condition gradually deteriorated and he died 12 hours postoperatively.

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Fig. 1a.



Fig. 1b.

Figs. 1a and b.—Radiographs taken with patient in (a) supine and (b) upright positions show the presence of free air in the peritoneal cavity.

An autopsy was performed and apart from the presence of a purulent peritonitis, the main findings of interest were noted on examination of the stomach. There was no leakage from the suture line in the stomach wall. After fixation of the stomach the sutures were removed and the tear and stomach wall examined thoroughly. The actual tear measured 2 cm. in length and adjacent to this there was an area of marked thinning of the stomach wall which measured 2.5 cm. in length and 1.2 cm. in width. The thin area transilluminated brightly.

Histologic examination revealed marked hypoplasia of the muscular coat of the stomach along the greater curvature (Figs. 2a and 2b, and 3a and 3b).

As an incidental finding, there was atresia of the proximal segment of the left ureter with a multicystic left kidney showing only a minimal amount of renal parenchyma.

Pathogenesis

Many ætiological factors have been shown to produce rupture of the stomach in the newborn infant, but our primary interest was in those cases which have shown a muscular defect in the stomach wall. Approximately 15 such cases have been reported.[1, 5, 6, 9-12, 19]

Acute or chronic peptic ulceration was the presumed cause of all gastric perforations in the newborn until 1943 when Herbut[6] demonstrated the presence of a muscle deficiency in the stomach wall, which he postulated to be the result of failure of fusion of the anlage of the circular muscle layers. While peptic ulcer is usually the cause of duodenal perforation in infants, it appears to account for less than half of the cases of gastric perforation. The ulcer that has been observed to accompany intracranial lesions has also been blamed for gastric perforation in infants,[3] but in most cases reported, no intracranial lesion has been present.

Septicæmia with gastric ulceration and necrosis of the stomach wall has resulted

in rupture of the stomach.[19] Some cases have been associated with distal obstruction in the gastrointestinal tract[7, 15] but not all of these have been associated with muscle deficiency in the stomach wall. Trauma to the abdomen[4] during delivery has been postulated as a cause but as yet is unproven. There is no air in the gastrointestinal tract until after birth and it would be a really traumatic delivery that ruptured a small non distended stomach. Perforation of the stomach by nasogastric tubes that were being used for aspiration or gavage feeding has also been reported.[19-21]

Perforation of gastric diverticula[2, 16] in the newborn period, with and without distal obstruction, have been reported. These cases can most likely be grouped with those showing congenital muscle defects since they exhibited a type of muscle inadequacy and were in the same location high on the greater curvature of the stomach.

Discussion

Gastric perforation in the newborn infant is an acute surgical emergency which requires prompt treatment. The clinical picture is remarkably constant and the case presented here is typical. The baby is usually well for the first few days of life. In the majority the sudden catastrophe occurs within the first week. Associated anomalies are often present and these may or may not contribute to the perforation. In our case there was an anomaly of the left ureter and kidney which obviously did not contribute to the rupture of the stomach.

When rupture occurs there may be a fairly rapid onset of abdominal distension with associated paralytic ileus, or, as in our case, this may be preceded by respiratory distress. It is suggested that the cause of this distress is irritation of the diaphragm, producing shallow, rapid, irregular breathing and poor air exchange in the lungs leading to the dyspnœa and cyanosis. This may well be a clue to diagnosis in the early stages. Radiological examination with the patient in the upright position will show free air in the peritoneal cavity indicating rupture of a hollow viscus and making laparotomy mandatory.

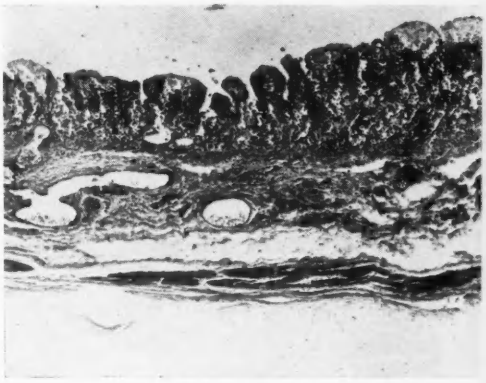


Fig. 2a.

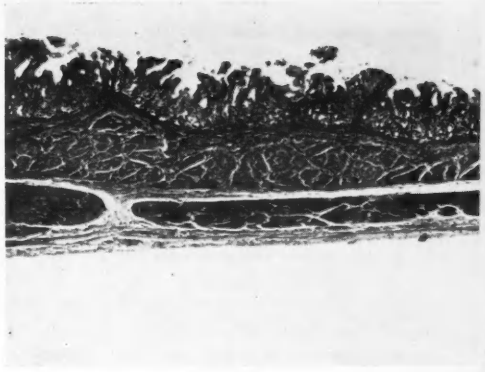


Fig. 2b.

Fig. 2a and b.—Photomicrographs of stomach wall (original magnification x 52). (a) From the case reported showing the markedly hypoplastic muscle layer and wide submucosa containing large vessels. (b) Normal stomach in the newborn infant, for comparison of thickness of muscle layer.

Pneumoperitoneum in the infant who continues to thrive has been treated conservatively,[18] but infants with gastric perforation are usually seriously ill and it is completely unjustified to delay operation no matter how tempting it may be to consider conservative treatment. Where there is a muscle defect, the tear in the stomach wall may be 1 cm. to 6 cm. in length and it is asking too much to expect it to seal spontaneously. Immediate surgical closure offers the only chance for survival. Abdominal paracentesis might help to relieve the respiratory distress but should not be allowed to delay operation.

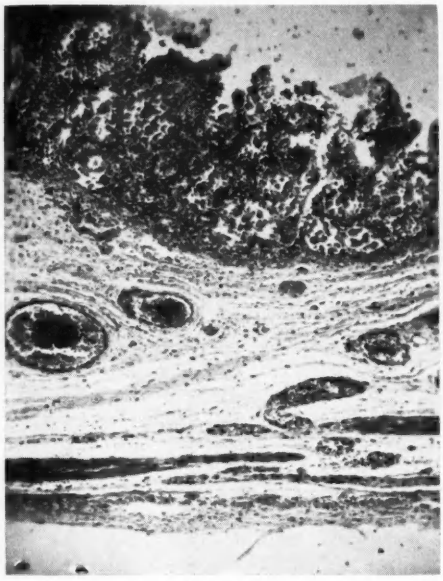


Fig. 3a.

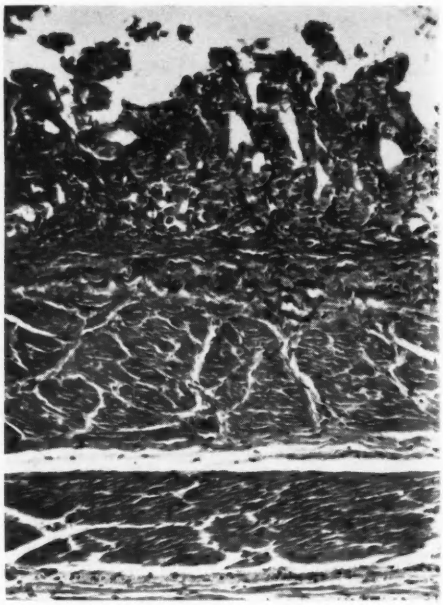


Fig. 3b.

Figs. 3a and b.—Photomicrographs of stomach wall (original magnification x 85). (a) From case reported. (b) From normal newborn infant's stomach. The higher magnification further stresses the pronounced lack of musculature in (a).

When closure is performed, the tear should be debrided and care taken to place sutures through healthy stomach wall. The debrided material will provide tissue for histological examination. Some of the reported surviving patients had no tissue submitted to the pathologist and, therefore, without casting any doubt on the clinical acumen of these authors, their diagnosis of congenital muscle defect must remain presumptive.[1, 10, 12]

The presence of the lesion high on the greater curvature is supposedly pathognomonic of congenital muscle defect but this is not necessarily true. Lesions in this location are not always associated with congenital deficiency in the musculature, and further, perforations in this location have occurred from other causes. Congenital muscle deficiency, however, is probably responsible for the vast majority of tears of the stomach wall high on the greater curvature and may well have been present in many cases previously attributed to peptic ulceration.

The results of operation can be improved. To date, the diagnosis of gastric perforation in the newborn has been made most often at postmortem examination. This however will change. Of the infants subjected to operation, the survival rate is approximately 30%. The chief cause of death is overwhelming peritonitis. Occasionally the associated anomalies play a part in the death of the infant. At the time of operation one must look for distal obstruction in the gastrointestinal tract. Successful treatment demands a suspicious mind, an early diagnosis and prompt surgical repair, if the peritonitis is to be controlled.

Summary

A case of spontaneous perforation of the stomach in a newborn infant is presented. The ætiology of the perforation is a congenital muscle deficiency in the stomach wall.

Perforation of the stomach in newborn infants is reviewed in general and that due to congenital muscle defects in particular.

Delay in diagnosis is the major factor at present responsible for the high mortality.

This is an acute surgical emergency in the newborn period and requires prompt surgical intervention if these patients are to have any chance of survival.

ACKNOWLEDGMENT

The authors extend their thanks to Dr. Ilona Perner, Assistant Pathologist of The Montreal Children's Hospital, for her assistance in the preparation of the pathological sections and the photomicrographs.

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RÉSUMÉ

La perforation gastrique chez le nouveau-né est rare. La première publication à ce sujet date de 1825 et est due à Siebold.[14] Depuis on en retrouve 70 dans la littérature. La première tentative de correction chirurgicale a été décrite par Stern[17] en 1929, tandis que la première survie est due à Legar en 1950. Le seul cas survenu au "Montreal Children's Hospital" est rapporté.

L'étiologie de la perforation était une déficience musculaire congénitale de la paroi gastrique postérieure haute du côté de la grande courbure. Le tableau clinique est assez constant et typique en ce que le calme des premiers jours de vie est soudainement brisé par le drame abdominal qui accompagne habituellement la perforation d'un organe creux qui se complique précocement de péritonite. La suture immédiate offre l'unique chance de survie.

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AN INTERESTING COMPLICATION OF JEJUNO-ILEAL DIVERTICULITIS

W. E. MYDLAND, M.D., F.R.C.S.[C], L. F. SPACKMAN, M.D. and V. T. MASON, M.D., *Calgary, Alta.*

Diverticula of the small bowel, exclusive of Meckel's diverticulum, are rare. They are usually encountered unexpectedly at operation or autopsy. Occasionally they are detected on barium examination of the upper gastrointestinal tract.

Incidence and Occurrence

They appear to occur more commonly in men, particularly in the 50 to 70 year age group. Edwards[3] found nine cases in 2820 autopsies, an incidence of 0.31%. This is probably closer to the true incidence than the estimates based on various reports of the occurrence of this lesion in radiological series. Shutkin,[1] Mahorner[2] and Ratcliffe[7] in their reports have discussed the incidence in detail and their observations need not be elaborated upon here, other than to say that these are the rarest of gastrointestinal tract diverticula. Diverticula of the gastrointestinal tract occur in the following sites in order of decreasing frequency: colon, ileum (Meckel's), duodenum, pharynx and œsophagus, stomach, and jejunum and ileum. Benson[6] mentions that they are frequently accompanied by congenital anomalies.

Pathology

Jejunal and ileal diverticula are of the pulsion type and are located along the mesenteric attachment. They are said to occur at sites where blood vessels perforate the bowel wall. The wall of such diverticula is made up of mucous membrane only, but there may be some stretched-out muscle fibres around the base. Edwards[3] has given a detailed description of their pathogenesis. The number and size of diverticula decrease distally in the small bowel from the ligament of Treitz to the ileocæcal valve. They may be large or small and may vary in number from a single diverticulum to several hundred.

Surgical Aspects

The surgeon becomes interested in this problem either because he encounters it at laparotomy or in his endeavours to treat complications resulting from the presence of diverticula. If these are found incidentally at laparotomy, most surgeons agree that they require no specific treatment. We feel that this is sound advice, firstly because they are predominantly asymptomatic and secondly because the excision of the involved bowel would have to be extensive, resulting in small bowel insufficiency. Pain, due to distension of the diverticulum, borborygmus, flatulence and vomiting have been attributed to their presence. A few cases in the literature would seem to substantiate this claim but the great majority appear to be asymptomatic. Complications of diverticulosis of the jejunum and ileum require more aggressive treatment.

The following complications have been encountered in our review of the available literature:[10, 11] acute or chronic mechanical intestinal obstruction, hæmorrhage,[5] inflammation,[4, 7] free perforation into the mesentery, pneumoperitoneum,[8] foreign bodies,[9] and carcinoma.[6] (A detailed classification of these has been recorded by Benson.[6]) That these complications do not occur more frequently is probably due to the fact that the diverticula have wide stomata which allow free drainage and mitigate against stomal obstruction.

Our present case presented a complication of diverticulosis ilei which has not been previously reported.

Case Report

J.S., a 79 year old white woman began having symptoms of intermittent suprapubic pain, urinary frequency and severe dysuria in 1958. She was treated unsuccessfully for urinary tract infection over the next six months. About January 1959 she noted some involuntary leakage of urine which she thought came from the vagina. She retained some urinary control. About one month before being seen at the Calgary Associate Clinic in August 1959, she noted slight vaginal bleeding.

Functional enquiry revealed that she was becoming weaker and that her weight had

decreased from 125 to 101 pounds. She had little appetite, occasional nausea and vomiting but no other gastrointestinal symptoms. She was having regular bowel movements with no melæna or bright red blood.

Physical examination revealed a rather frail old lady, who appeared to be her stated age of 79 years. The chest was clear to auscultation. Cardiac examination revealed normal rhythm with heart sounds of good quality, no murmurs and a blood pressure 150/100. No masses, enlarged organs, tenderness or rigidity were noted on abdominal examination. Pelvic examination was unsatisfactory but revealed a stenosed vaginal vault. Rectal examination was negative.

The patient was admitted to the Holy Cross Hospital, Calgary, on August 31, 1959. Under spinal anæsthesia a cystoscopic examination was performed and the urologist noted a fistulous opening on the floor of the bladder on the right side, just above the right ureteral orifice. Fæcal material could be seen entering the bladder through this opening. He felt that this did not have the appearance of a malignant fistula. A cystogram was then done which showed filling of the terminal ileum and right colon. Diverticula could be seen along the bowel wall and these were erroneously interpreted as being of colonic origin. Plain films of the abdomen showed calcified gallstones (Figs. 1 and 2).

While the patient was still under anæsthesia, a pelvic examination was carried out and this revealed senile vaginal changes associated with stenosis of the upper third of the vagina. The gynæcologist also felt that there was a cystic mass about three inches in diameter in the pouch of Douglas. Definition of other pelvic structures was impossible. It was the gynæcologist's opinion that the mass was of inflammatory origin.

On the basis of the above information it was felt that an abdominal exploration should be carried out. Accordingly, on September 14, 1959 after preparation of the bowel with neomycin, laparotomy was performed. This revealed diverticulosis of the jejunum and ileum. A loop of terminal ileum was adherent to the right side of the bladder. A soft cystic mass lay in the pouch of Douglas. The rest of the pelvis was remarkably free of adhesions or other signs of disease. The gallbladder was adherent to the duodenum, omentum and hepatic flexure of the colon. The terminal ileum was first separated from the bladder by blunt dissection. This revealed the opening into the bladder which was then closed. The portion of the ileum with the diverticulum which had formed the fistula, was resected and end to end anastomosis was performed. The cystic mass proved to be a serous cyst of the right ovary. This was excised together with the right Fallopian tube. Following this the gallbladder was mobilized to be certain there was no cholecystenteric fistula. Large stones could be palpated through its wall. The patient had tolerated the procedure very well to this point, so cholecystectomy was performed.

The patient did reasonably well postoperatively, except for a period of electrolyte imbalance which was moderately difficult to control. She was discharged from hospital on October 4, 1959, and was eating and voiding well. Bowel movements were normal and urinalysis showed that the urine was clear. Urinary control was excellent.

Pathological Report

The specimen consisted of a portion of ileum 32 cm. long, containing a number of

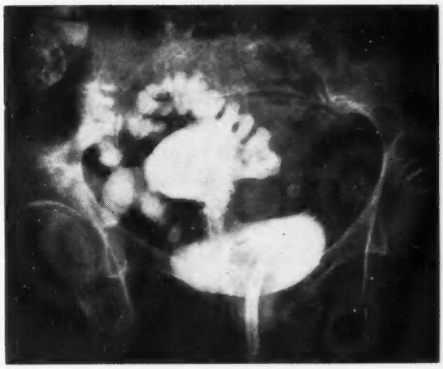


Fig. 1.



Fig. 2.

Figs. 1 and 2.—Cystograms demonstrating ileovesical fistula.

diverticula and in one area showing perforation with an inflammatory reaction surrounding it. No foreign body was present although a fæcolith was present at the site of the fistula into the bladder. A cyst 11 cm. in diameter, with attached Fallopian tube was present. This was a simple serous cyst. The third specimen was a gallbladder containing four calculi and showing the changes of chronic cholecystitis.

Treatment

Medical treatment[7] of small bowel diverticulosis consists of low residue diets, antispasmodics, mild laxatives and postprandial postural drainage in the prone position. This programme has been of some benefit to those without complications but is of no value to those with complications.

Surgical treatment has consisted of closure of perforations, inversion of diverticula,[7] enteroanastomosis to bypass areas of diverticulosis,[6] and resection with reestablishment of intestinal continuity,[2, 4, 8] The latter appears to be the procedure of choice provided sufficient small intestine is left to carry on adequate absorptive function. In patients with total involvement of the small bowel, as in the case reported here, only a short segment containing the involved diverticulum should be resected with reestablishment of bowel continuity.

Summary

A general discussion of jejuno-ileal diverticula is given with a list of commonly encountered complications. To this is added a case report of an ileo-vesical fistula resulting from diverticulitis of an ileal diverticulum.

Recommended treatment is discussed in general terms.

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Résumé

Les diverticules du grêle, le Meckel exclus, sont rares. Edwards[3] en a trouvé neuf cas au cours de 2820 autopsies, soit une incidence de 0.31%.

Ils sont habituellement asymptômatiques et ne requièrent aucun traitement. Ils deviennent d'intérêt chirurgical lorsqu'ils se compliquent. Le cas rapporté est celui d'une femme de 79 ans qui commença d'accuser douleur intermittente suspubienne, de la pollakyurie et une dysurie importante. Traitée pour infection urinaire sans succès pendant 6 mois, elle nota alors une certaine incontinence urinaire, puis accusa, un mois avant d'être vue au "Calgary Associate Clinic", de l'écoulement vaginal sanguinolent.

Admise au "Holy Cross Hospital", elle subit, sous rachi-anesthésie, une cystoscopie qui démontra un orifice fistuleux duquel s'écoulaient des débris fécaloïdes. Un cystogramme "visualisa" l'iléon terminal et le colon droit et montra des diverticules.

Une laparotomie confirma la présence d'un diverticule iléal fistulisé dans la vessie. La perforation vésicale fut suturée et un segment d'iléon de 32 cm., qui comprenait d'autres diverticules, réséqué. L'opération fut complétée d'une salpingo-ovariectomie droit et d'une cholécystectomie.

SURGICAL TECHNIQUE

TABLE FOR SURGERY OF THE HAND

W. D. BUTT, M.D.,* *Toronto*

THERE HAVE BEEN many tables designed to aid in surgery of the hand. The most common of these is simply a separate table on wheels beside the operating table. The difficulty is that the two tables become separated with any motion on the part of the surgeon or his assistant, and the draping may become unsterile.

Commonly a large arm board is placed under the mattress. This is unstable when the patient is moved and there is no support under the hand during the operation.

This table† is designed with the following features:

1. The legs can be folded and it may be transported easily.
2. The operating surface is easily cleaned.
3. It is attached to the side bar of the operating table so that it must move with the operating table as a unit.
4. It may be rotated on an eccentric axis so that the hand table may be parallel with or at right angles to the operating table, or in any position which is most convenient to the surgeon.
5. The table may be rotated out of the way and the hand thoroughly prepared while the arm is extended over a floor bucket or basin. The table is then draped and rotated under the freshly prepared hand and arm so that complete sterility is maintained. Details of the hand table are depicted in the illustration. The photograph shows the surgeon, assistant and nurse, and the draped table and Mayo stand. The prepared hand is ready for surgery.

RÉSUMÉ

Plusieurs tables ont été proposées pour favoriser la chirurgie de la main à partir de la table séparée, sur roulettes, qui a l'inconvénient de se déplacer trop facilement, jusqu'à la planchette retenue sous le matelas de la table d'opération, qui donne vraiment un support insuffisant.

Une table qui serait retenue d'un côté sous le matelas et soutenue de l'autre par des pattes, semblerait idéale. Elle offre comme avantages:

1. Des pattes qui peuvent se basculer, ce qui facilite son déplacement.
2. Une surface qui peut être nettoyée facilement.
3. D'être fixée à la barre de côté de la table d'opération de sorte qu'elle constitue l'unité de mouvement.
4. De se mouvoir selon un axe excentrique afin d'offrir les angles les plus utiles selon les cas.
5. De pouvoir être écartée au besoin, lors de la préparation de la main, et remise en place après mise des champs pour recevoir le membre.

Les détails sont illustrés.

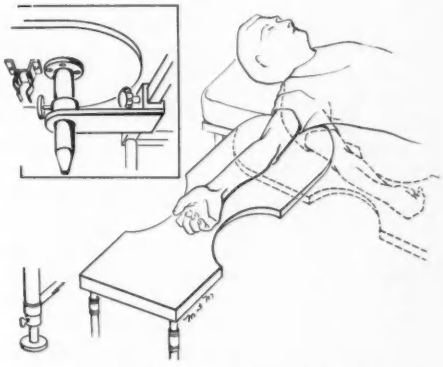


Fig. 1.—Detailed diagram of hand table.



Fig. 2.—Draped table and Mayo stand. The prepared hand is ready for surgery.

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EXPERIMENTAL SURGERY

VARIATIONS PHYSIOLOGIQUES AU COURS DE LA CIRCULATION EXTRA-CORPORELLE DANS LE LABORATOIRE*

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LA CRÉATION d'un service de chirurgie cardio-vasculaire dans un hôpital général de l'envergure de l'Hôtel-Dieu St-Vallier entraîne certaines exigences tant du côté administratif que médical.

Ce service nécessite en effet pour fonctionner des locaux et un matériel très onéreux et une collaboration étroite avec la plupart des disciplines de l'Hôpital, en particulier des services de cardiologie, de radiologie, d'anesthésie, et du laboratoire. Mais en outre un service de chirurgie cardiaque ne se conçoit pas sans un laboratoire de recherches expérimentales. C'est dans de tels laboratoires qu'a commencé cette chirurgie, c'est là où se réalisent les progrès, où s'élaborent les techniques opératoires nouvelles et se crée la sécurité opératoire.

Ce service est né il y a un an à peine. La première circulation extra-corporelle totale expérimentale a eu lieu le 2 février 1959; la première circulation extra-corporelle humaine chez un enfant de six ans ayant une communication inter-ventriculaire a eu lieu le 12 novembre 1959 avec succès.

S'engager dans cette discipline chirurgicale cinq ans après le début de la chirurgie à cœur ouvert offre déjà un avantage sensible car le chirurgien peut ainsi bénéficier de l'expérience de centres réputés et travailler avec un matériel éprouvé et moderne, évitant ainsi la période d'essai et de tâtonnement longue et onéreuse.[1-5]

Le travail aujoud'hui présenté, est le résultat des 30 prémières circulations extra-corporelles totales dans le laboratoire. En pratiquant ces 30 circulations extra-corporelles, l'objectif ultime n'était pas tellement d'essayer de faire avancer la chirurgie cardiaque mais de mettre au point cette chirurgie dans le laboratoire et de comparer les résultats obtenus avec les résultats de centres d'avant-garde, avant de transposer cette chirurgie à l'humain. En chirurgie cardiaque, et ceci s'applique à toute chirurgie, le succès dépend de l'honnêteté des résultats de l'expérimentation.

Ces 30 circulations extra-corporelles ont permis d'éprouver d'une façon sûre:

1. l'appareil cœur-poumon-artificiel.
2. la compétence et l'habileté des techniciens.
3. l'entraînement des infirmières.
4. les différentes techniques opératoires actuellement au point pour la correction des lésions cardiaques.
5. l'efficacité du laboratoire et de l'anesthésie.

Ces 30 circulations comprennent toutes celles qui ont été pratiquées entre le 2 février 1959 et le 23 juin 1959. Il n'y a pas eu de période d'essai ou de circulations rejetées. Les résultats sont exposés en photos, tableaux, graphiques et commentaires et celà avec la plus grande objectivité.

Le système pompe-oxygénateur employé a été le système Dewall-Lillehei (Fig. 1): pompe à doigts sigma-moteur et oxygénateur à bulles. L'oxygénateur (Fig. 2) est constitué d'un tube en mayon d'un pouce ½ de diamètre par 65 cm. à 75 cm. de long; par l'extrémité inférieure de ce tube arrivent le sang et l'oxygène. Le sang arrive par une seule ouverture centrale entourée d'au moins 150 ouvertures minuscules pour l'oxygène. L'extrémité supérieure est trouée sur 10 cm. Ces trous ont 1 cm. de diamètre; le sang est démoussé à ce niveau par des éponges de Stain-o-steel nettoyées par un procédé à la soude caustique et imbibées de Silicone traité à l'éther. Le sang retombe sur un filtre en forme de mouchoir. Cet ensemble (tube, éponges, filtre) est placé à l'intérieur d'une "Canister" ayant à sa partie inférieure deux

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*Travail effectué grâce à la collaboration des autorités de l'Hôtel-Dieu St-Vallier de Chicoutimi.
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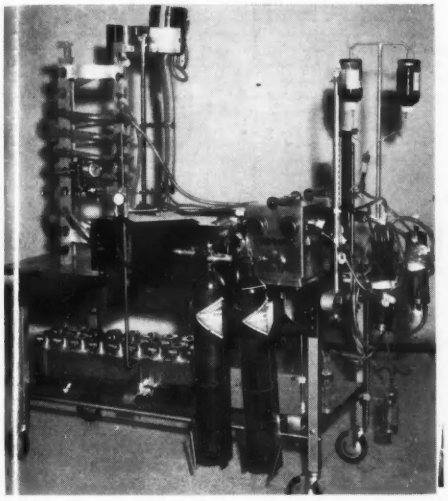


Fig. 1.—Photo de l'appareil cœur-poumon artificiel tel qu'employé à l'Hôtel-Dieu St-Vallier.

sorties, une pour le niveau du sang et l'autre pour l'écoulement du sang oxygéné dans le serpentin. Le sang sort ainsi de cet ensemble démoussé et débarrassé de débris tels que: caillots, fibrine, silicone. Le serpentin constitue le réservoir artériel où la pompe sigma-moteur artérielle puise le sang pour le retourner à l'opéré.

Ce système pompe-oxygénateur comprend d'autre part la succion de cardiotomie qui aspire sous 15 mm. de Hg. le sang à l'intérieur du cœur. Il peut y avoir deux succions de cardiotomie et une autre succion fonctionnant par gravité; celle-ci est employée lors de la canulation de l'oreillette gauche. Cette canulation est importante, surtout dans les gros retours des artères bronchiques et les arrêts cardiaques afin d'éviter la surdistension du cœur gauche et du lit pulmonaire et au besoin pour aspirer les bulles d'air emprisonnées dans l'oreillette gauche et menaçant de franchir l'aorte et ses branches.

Les chiens utilisés ont tous été des chiens bâtards récupérés dans la région. Leur poids (Tableau I) a varié de 8.650 kg. à 31 kg., avec une moyenne de 15.526 kg. Chaque animal, une heure avant d'être anesthésié reçoit morphine 1/6 gr. et atropine 1/150 gr. L'anesthésie est faite uniquement avec du pentothal intraveineux à 50 mg. au c.c. L'anesthésie est superficielle. Le chien anesthésié et intubé respire par lui-même jusqu'à l'ouverture du thorax. A ce moment, la respiration est contrôlée à l'aide d'un appareil automatique sous air comprimé. L'air est de beaucoup préférable, car l'oxygène pur chasse l'azote du sang et augmente la pression partielle de l'oxygène dans le sang. L'artère fémorale gauche est toujours canulée avec un cathéter de polyéthylène et la pression prise de façon continue sur un appareil à mercure et périodiquement sur papier enregistreur. Les tracés électrocardiographiques de même que l'électro-encéphalogramme sont contrôlés continuellement au moyen d'un visoscope et à certains moments ils sont également enregistrés sur papier (Figs. 3 et 4). La température rectale est prise à volonté à l'aide du thermocouple et le chien repose sur le matelas de la machine therm-o-rite. Ainsi sont créés des conditions exactement semblables à celles exigées dans la salle d'opération humaine. Nous



Fig. 2.—Ensemble oxygénateur et chambre de démoussage. Tube de mayon, éponges de Stain-o-steel, filtre, "canister".

nous servons toujours du thermocautère. Le "ground" du thermocautère chez le chien est obtenu en plantant une aiguille dans une patte-arrière.

Nous n'avons jamais pratiqué de groupes sanguins chez les chiens. La mortalité par incompatibilité sanguine chez le chien étant de l'ordre de 10%, les accidents sont rares et les chirurgiens de la plupart des centres reconnus ne procèdent pas au groupement sanguin des animaux. Autant que possible cependant, nous choisissons un chien donneur ayant des caractères physiques se rapprochant le plus possible de l'animal à opérer.

Le chien donneur, suivant son poids, reçoit de 4 à 7 mg. de succinylcholine. Cette dose n'est jamais dépassée. Cette posologie a toujours été suffisante et efficace pour au moins 20 minutes. Il est ensuite intubé et hyperoxygéné. Le sang est prélevé au niveau de l'artère fémorale dans des bouteilles siliconées de 500 c.c. contenant 18 mg. d'héparine et 30 c.c. de soluté glucosé à 5%. La plupart des chiens donneurs sont conservés vivants en leur injectant 1000 c.c. de soluté glucosé à 5% ou de soluté salé physiologique par la veine fémorale. Ils peuvent ainsi être utilisés à nouveau comme donneur après une période d'un mois de repos.

Le sang prélevé est conservé au bain-marie à 37° c. jusqu'à son utilisation dans le cœur-poumon artificiel.

Tous les chiens opérés ont eu une thoracotomie droite dans le 5ième espace intercostal avec hémostase soignée. L'héparine a été donnée à la dose de 1.5 mg. par kilogramme de poids. La veine cave inférieure a été canulée par l'auricule droite et la veine cave supérieure, par la veine azygos. L'artère carotide droite a été canulée avec un cathéter de plus gros calibre possible introduit sur 2 à 3 cm.; on ne le pousse jamais plus loin pour ne pas obstruer la bifurcation carotidienne. A la fin de la circulation, la protamine a été injectée à la dose de 3 mg. par kilogramme de poids.

Le débit du cœur-pouman artificiel (Tableau I) a été de 55 c.c. à 90 c.c./kilogramme/minute avec une moyenne de 72.5 c.c./kilogramme/minute ou de 1112 c.c./minute en moyenne. La pression avant

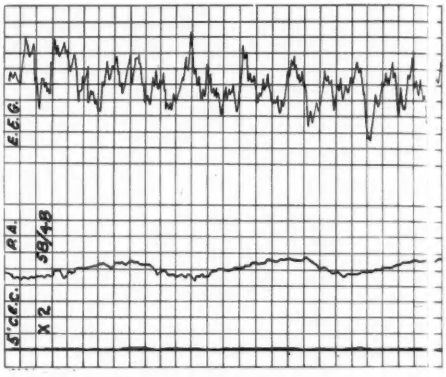


Fig. 3.—Electro-encéphalogramme et pression artérielle (58/48) cinq minutes après le début de la circulation extra-corporelle.

circulation mesurée dans la fémorale était de 125 systolique en moyenne et a varié de 60 à 110 systolique durant la circulation, la moyenne s'établissant à 73 systolique (Fig. 3). La durée de la circulation extra-corporelle totale a varié de 28 minutes à 101 minutes et 15 secondes avec une durée totale de 2004 minutes et 24 secondes et une moyenne de 66 minutes et 48 secondes pour chaque circulation.

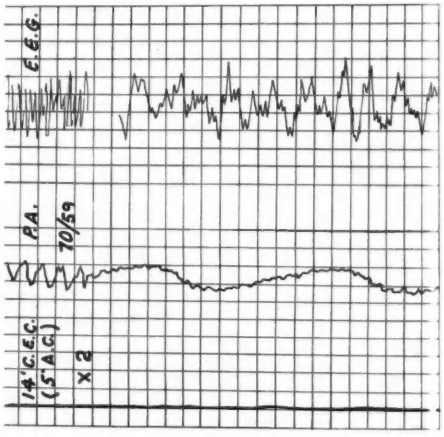


Fig. 4.—Electro-encéphalogramme et pression artérielle (70/54) 14 minutes après le début de la circulation extra-corporelle et cinq minutes après l'arrêt cardiaque.

Avant d'endormir le chien qui aura une circulation extra-corporelle, nous prélevons environ 1500 c.c. de sang chez un chien assez gros (de 60 à 120 livres environ). Ce sang servira à remplir le cœur-poumon artificiel.

TABLEAU I

N° de l'expér.	Date 1959 quant.	mois	N° du chien	Poids de l'animal en kg.	Débit Total en c.c.	c.c./kg.	Pression en mm. Hg. avant	durant	Température en °C avant	après	Durée de la perfusion min.	sec.	VD	PVD	K (Durée en min.)	AD	Saaa	Cog	Ag	AAA	Temps en heures
1	2	2	15	16	960	60	130	80	37.5	35.8	31		+								2
2	6	2	21	17	1020	60	145	90	37.6	35.2	55		+								1
3	10	2	25	24.5	1440	60	140	80	37.5	36.3	28		+	+							
4	12	2	31	15.5	930	60	140	80	37.2	35.7	37		+	+	12′						
5	23	2	29	13.5	900	65	130	80	34.7	32.4	43		+	+	11′						4
6	23	2	16	12.1	780	65	120	70	38.4	38.5	65	30	+	+							
7	27	2	19	13.05	910	70	110	80	38.5	36.7	60		+	+	11′						
8	3	3	39	9.05	630	70	120	60	37.8	36.7	68		+		21′		+				
9	5	3	10	15.30	1050	70	120	60	38.9	36.5	63	30	+		12′	+	+				
10	9	3	26	11.85	720	60	125	65	37.2	35	60	30	+	+							2
11	11	3	41	8.65	720	80	150	50	39.3	36.6	75		+	+							2
12	13	3	35	12.45	1000	80	150	80	37.3	35.9	76		+	+		+					1
13	17	3	42	10.65	880	80	160	95	37.8	35	94	30	+	+		×2			+		
14	19	3	45	16.85	1350	80	105	60	38.6	36.5	84	10	+	+							2
15	1	4	44	22.50	1840	80	135	110	37.8	34.8	78		+	+							2
16	6	4	51	21.75	1760	80	125	80	34.5	38	65		+			+			+		2
17	8	4	48	17.85	1440	80	140	70	38.6	35.2	64		+		22′					+	2
18	13	4	22	31.00	2170	70	100	40	39.7	37.5	65		+		15′			+			
19	15	4	56	14.50	1200	80	130	100	37.9	36.5	67		+	+							½
20	28	4	65	15.25	900	60	130	60	37.6	34.5	61		+								1
21	1	5	64	11.425	990	90	120	70	37.6	33.5	101	15	+	+				+			1
22	5	5	63	23.50	1380	60	135		37.8	35.6	65	20	+	+							2
23	12	5	57	10.925	935	85	95	55	37.6	35.6	83	20	+	+		+					3
24	15	5	75	9.80	750	75	95	90	36.6	34.3	90	10	+	+		+					1
25	20	5	40	12.45	960	80	100	80	36	35	55	50	+	+							2
26	10	6	89	19.40	1200	60	120	60	38.3	34.5	80	20	+	+							
27	16	6	83	19.70	1400	70	160	120	36.2	34.5	71	20	+	+		+					
28	17	6	76	15.35	1200	80	110	75	37.5	34.2	73	10	×2	×2		+					3
29	19	6	77	10.45	850	85	110	75	35.7	34	71	20	×2	×2							3
30	23	6	78	14.00	1120	80	120	75	36.6	33.8	72	12	+	+							2
Moyenne				15.526	1112.83	72.5	125.6	73	37.476	36.66	66	48									

LÉGENDE: VD: Ventriculotomie droite. PVD: Patch ventriculaire droit. K: Arrêt cardiaque. AAA: Aortotomie de l'aorte ascendante. AD: Auriculotomie droite. AG: Auriculotomie gauche. Saaa: Section de l'aorte ascendante–anastomose. Cog: Canulation de l'oreillette gauche. AV: Avant. D: Durant. AP: Après.

La température du chien avant circulation était en moyenne de 37.4° c. et de 36.6° c. à la fin de la circulation.

Au cours de ces 30 circulations extracorporelles il a été pratiqué:

Ventriculotomie droite:	32
"Patch" infundibulaire droit:	24
Section et anastomose de l'aorte ascendante:	3
Auriculotomie droite:	8
Auriculotomie gauche:	2
Canulation de l'oreillette gauche:	2
Arrêt cardiaque:	7

Il y a 32 ventriculotomies droites car deux fois il y a eu ventriculotomie sur la chambre de chasse et ventriculotomie sur la chambre de remplissage du ventricule droit, puis mise en place de deux patchs sur ce ventricule. Les arrêts cardiaques ont été produits avec du citrate de potassium à 25% (2 c.c. dans 18 c.c. de sang), suivant le principe de Melrose; ou encore à l'acétylcholine, introduisé pour la première fois par Lam de Détroit. Ces arrêts ont varié de 11 minutes à 22 minutes, mais cette technique ne nous a pas satisfaits. C'est dans ces cas que nous avons eu de la difficulté à maintenir une pression normale et les chiens 29 et 10 sont morts de nécrose cardiaque. Actuellement nous avons complètement abandonné ces méthodes d'arrêt cardiaque. Nous utilisons présentement une solution électrolytique[8] froide qui nous donne des survies définitives avec arrêt cardiaque au-dessus de 50 minutes. Les suites opératoires ont été bonnes et le chien a marché très tôt après l'opération (Tableau I). Le tube thoracique enlevé dans la soirée avait drainé en moyenne 50 à 100 c.c. de sang.

Le tableau II indique la survie, les incidents au cours de l'opération et les causes de la mort.

Nous attribuons une mort, celle du chien N° 25, au cœur-poumon artificiel: le démoussage inefficace a entraîné des pertes de sang considérables et la pénétration de 200 c.c. d'air dans le circuit artériel. Nous avons enregistré un mauvais démoussage à quatre reprises soit: pour les chiens N°s 40-89-83-78 sans conséquence cependant.

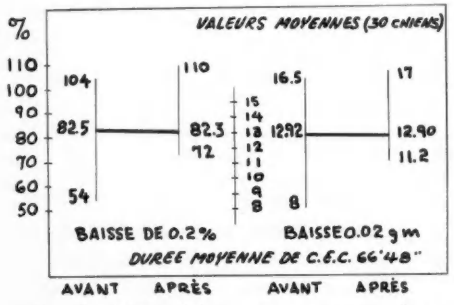


Fig. 5.—L'hémoglobine était avant circulation extra-corporelle de 82.5% ou 12.92 gm. en moyenne avec maximum de 104% = 16 gm. 5 et minimum de 54% = 8 gm. et après de 82.3% = 12.90 gm. en moyenne avec maximum de 110% = 7 gm. et minimum de 72% = 11.2 gm. donc une baisse moyenne de 0.2% = 0.02 gm.

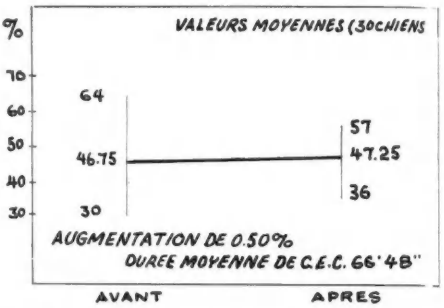


Fig. 6.—L'hématocrite était avant circulation extracorporelle de 46.75% de moyenne avec maximum de 64% et minimum de 30% et après de 47.25% de moyenne avec maximum de 57% et minimum de 36% c'est-à-dire qu'il a été enregistré une augmentation moyenne de 0.50%.

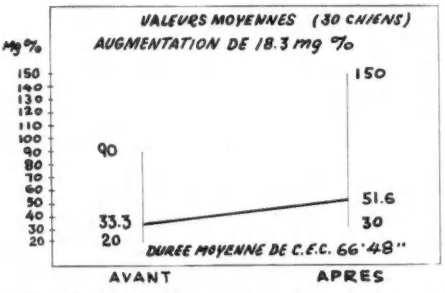


Fig. 7.—L'hémoglobine plasmatique était avant circulation extra-corporelle de 33.3 mg. pour 100 c.c. de moyenne avec maximum de 90 mg. et minimum de 20 mg. et après de 51.6 mg. pour 100 c.c. de moyenne avec maximum de 150 mg. et minimum de 30 mg. c'est-à-dire une augmentation moyenne de 18.3 mg. pour 100 c.c. de sang et le chiffre d'hémolyse le plus élevé n'a pas été enregistré dans la circulation la plus longue, car celle de 101' et 15" avait seulement 80 mg. d'hémoglobine plasmatique après circulation.

TABLEAU II

N°	Date 1959 quantième	mois	N° du chien	Durée de la perfusion min.	sec.	Incidents au cours de la circulation	Survie	Causes de la mort
1	2	2	15	31			Vivant	
2	6	2	21	55			Vivant	
3	10	2	25	28				La chambre de démoussage ne fonctionne pas, finalement 200 c.c. d'air pénètre dans système artériel.
4	12	2	31	37		Patch trop perméable	16J	Insuffisance cardiaque (ascite-pleurésie); il avait eu arrêt cardiaque.
5	23	2	29	43		Ouverture de l'aorte ascendante perte de 400 c.c. de sang	15J	Œdème aigu du poumon (nécrose du V.G.); il avait eu arrêt cardiaque.
6	25	2	16	65	30	Hémorragie au niveau de l'aorte ascendante postérieure.	Vivant	
7	27	2	19	60			Vivant	
8	3	3	39	68		Hémorragie au niveau de l'anastomose postérieure		Hémorragie sur l'aorte ascendante postérieure à la suite d'arrêt cardiaque; manque de sang.
9	5	3	10	63	30			Nécrose cardiaque.
10	9	3	26	60	30		Vivant	
11	11	3	41	75			Vivant	
12	13	3	35	76		Petit trou sur la face post - externe de la V.C.S. (suture avec soie 000000)	7J	Insuffisance cardiaque (pleurésie hémorragique): patch trop large.
13	17	3	42	94	30	Bloc auriculo - ventriculaire	1J	Bloc auriculo-ventriculaire par hématome septal auriculaire.
14	19	3	45	84	10		Vivant	
15	1	4	44	78			16J	Pneumothorax par plaie ouverte.
16	6	4	51	65			119J	Sacrifié.
17	8	4	48	64		Turbulance dans le tube d'oxygénation	5J	Insuffisance cardiaque (il avait eu arrêt cardiaque).
18	13	4	22	65			4H	Hémorragie (vraisemblablement par incompatibilité sanguine).
19	15	4	56	67			21J	Œdème du poumon. Epanchement pleural.
20	28	4	65	61		Turbulance dans le tube d'oxygénation	40J	Diarrhée.
21	1	5	64	101	15		Vivant	
22	5	5	63	65	20	Turbulance dans le tube d'oxygénation	25J	Abcès péricardique. Œdème du poumon.
23	12	5	57	83	20		Vivant	
24	15	5	75	90	10		Vivant	
25	20	5	40	55	50	Mauvais démoussage	Vivant	
26	10	6	89	80	20	Mauvais démoussage	8H	Hémorragie. Incompatibilité sanguine?
27	16	6	83	71	20	Mauvais démoussage panne de courant: 1′ 30″	3J	Sacrifié: décérébré
28	17	6	76	73	10		7J	Sacrifié (insuffisance cardiaque) Ascite-épanchement pleural.
29	19	6	77	71	20		25J	Sacrifié (ascite-épanchement pleural).
30	23	6	78	72	12	Démoussage médiocre	Vivant	

LEGENDE.—H-heures: J-jours.

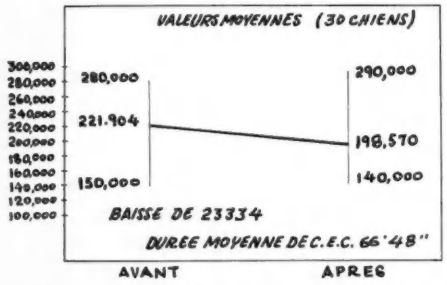


Fig. 8.—Les plaquettes sanguines étaient avant circulation extra-corporelle de 221,904 en moyenne avec maximum de 280,000 et minimum de 150,000 et après de 198,570 en moyenne avec maximum de 290,000 et minimum de 140,000, c'est-à-dire une baisse moyenne de 23,334.

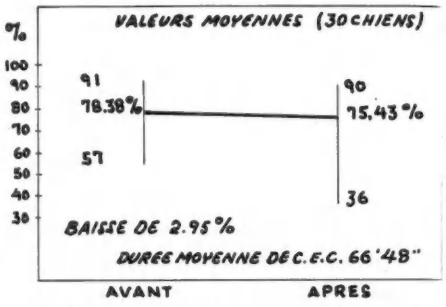


Fig. 10.—Les polynucléaires neutrophiles étaient avant circulation extra-corporelle de 78.38% en moyenne avec maximum de 91% et minimum de 57% et après de 75.43% en moyenne avec maximum de 90% et minimum de 36% c'est-à-dire une baisse moyenne de 2.95%.

La cause en a été retrouvée: les éponges de Stain-o-steel étaient stérilisées huit jours à l'avance et le silicone était devenu sec et inefficace. Une turbulence un peu trop énergique dans le tube d'oxygénation a été remarquée trois fois (chiens 48-65-63) sans conséquence. Il y a eu une panne de courant d'une minute et 30 secondes, (chien N° 83). La pompe s'étant arrêtée au moment où le cœur était ouvert, le chien est demeuré ensuite décérébré et a dû être sacrifié trois jours plus tard. Cet accident nous a incité à faire installer en chirurgie cardiaque humaine une unité spéciale d'urgence pour la salle d'opération humaine. Elle consiste en un dynamo fonctionnant à l'essence. Nous sommes convaincus qu'un système d'urgence pour tout l'hôpital n'est pas suffisant, car il ne règle pas le problème de la panne locale qui demande toujours un certain temps pour être réparée. Or en chirurgie à cœur-ouvert, une minute ou deux minutes de panne de courant peuvent être fatales pour l'opéré. Au cours de la création d'une communication interauriculaire et de sa réparation, il s'est formé un hématome sur la région du nœud de Tawara (chien N° 42) et celui-ci est mort 24 heures plus tard malgré l'installation du pace-maker artificiel. Les chiens 89 et 22 sont morts quatre heures et huit heures après l'opération. Nous avons attribué ces morts à une incompatibilité sanguine. En effet ces chiens ont présenté un saignement incontrôlable.

Nous avons pu conserver vivants 12 chiens ayant des "patches" sur la région infundibulaire du ventricule droit. Ces animaux sont en excellente santé et pério-

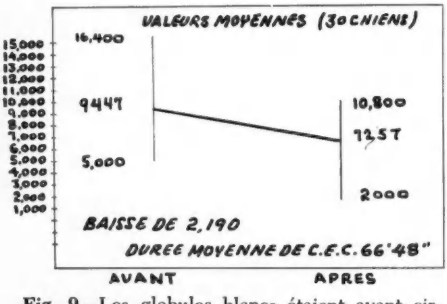


Fig. 9.—Les globules blancs étaient avant circulation extra-corporelle de 9447 en moyenne avec maximum de 16,400 et minimum de 5000 et après de 7257 en moyenne avec maximum de 10,800 et minimum de 2000, c'est-à-dire une baisse moyenne de 2190.

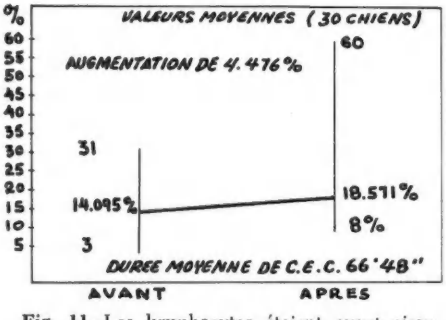


Fig. 11—Les lymphocytes étaient avant circulation extra-corporelle de 14.095% en moyenne avec maximum de 31% et minimum de 3% et après de 18.57% en moyenne avec maximum de 60% et minimum de 8%, c'est-à-dire une augmentation moyenne de 4.47%.

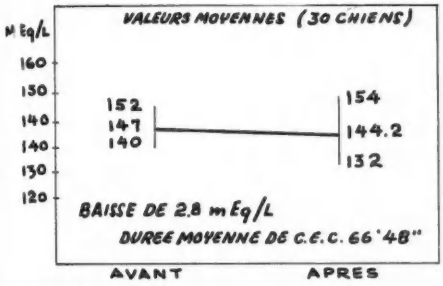


Fig. 12.—Le sodium artériel était avant circulation extra-corporelle de 147 mEq./l. en moyenne avec maximum de 152 et minimum de 140 et après circulation de 144.2 mEq./l. en moyenne avec maximum de 154 et minimum de 132, donc une baisse moyenne de 2.8 mEq./l.

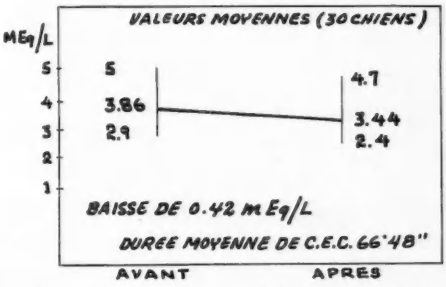


Fig. 14.—Le potassium artériel était avant circulation extra-corporelle de 3.86 mEq./l. en moyenne avec maximum de 5 et minimum de 2.9 et après circulation de 3.44 mEq./l. avec maximum de 4.7 et minimum de 2.4 donc une baisse moyenne de 0.42 mEq./l. en moyenne.

diquement, ils seront l'objet d'étude hémodynamique et cinéangio-cardiographique. S'ils meurent éventuellement, une étude anatomique complète en sera faite.

La série simplifiée de graphiques qui va suivre indiquera les résultats hématologiques et biochimiques pratiqués avant et après les circulations extra-corporelles.[6, 7] Les résultats présentés proviennent de moyenne établie sur ces 30 chiens. Les résultats sont également basés sur une moyenne de 66 minutes et 48 secondes de circulation extra-corporelle totale pour chaque chien.

Chaque critère de l'hémogramme complet est analysé. La formule sanguine comprend:

Sang Arteriel
L'hémoglobine,
L'hématocrite,
L'hémoglobine plasmatique,
La leucocytose,
Les polynucléaires neutrophiles,
Les lymphocytes,
Les plaquettes sanguines.

Les éléments biochimiques analysés sont les suivants:

Sang Arteriel
L'oxygène,
Le CO_2,
Le PCO_2,
Le pH,

Sang Veineux
L'oxygène,
Le CO_2,
Le PCO_2,
Le pH,

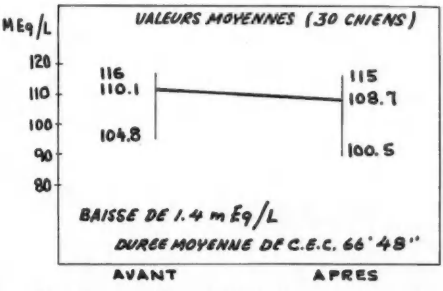


Fig. 13.—Le chlore artériel était avant circulation extra-corporelle de 110.1 mEq./l. en moyenne avec maximum de 116 et minimum de 104.8 et après circulation de 108.7 mEq./l. en moyenne avec maximum de 115 et minimum de 100.5 donc une baisse moyenne de 1.4 mEq./l.

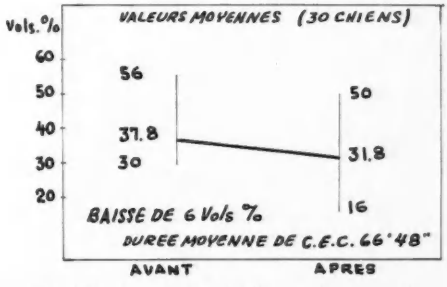


Fig. 15.—La réserve alcaline était avant circulation extra-corporelle en moyenne de 37.8 vols % avec maximum de 56 et minimum de 30 et après circulation de 31.8 vols % en moyenne avec maximum de 50 et minimum de 16 donc une baisse moyenne de 6 vols %.

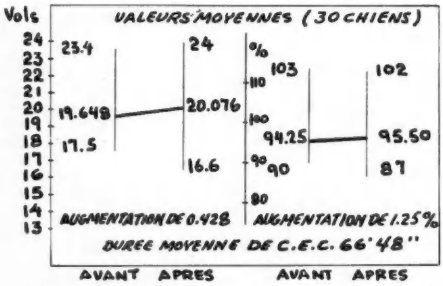


Fig. 16.—L'oxygène artériel était au début de la circulation extra-corporelle de 19.648 vols ou 94.25% en moyenne avec maximum de 23.4 vols ou 103% et minimum de 17.5 vols ou 90% et à la fin de la circulation de 20.076 vols ou 95.50% en moyenne avec maximum de 24 vols ou 102% et minimum de 16.6 vols ou 87% donc une augmentation moyenne de 0.428 vols ou 1.25%.

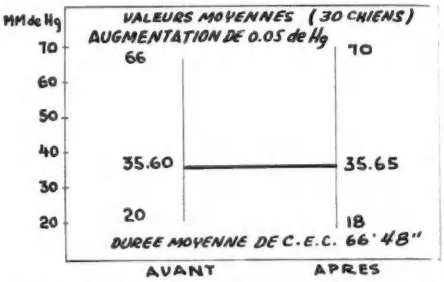


Fig. 18.—Le PCO_2 artériel était au début de la circulation extra-corporelle de 35.6 mm. de Hg en moyenne avec maximum de 66 et minimum de 20 et à la fin de la circulation de 35.65 mm. de Hg en moyenne avec maximum de 70 et minimum de 18 donc une augmentation moyenne de 0.05 mm. de Hg.

SANG ARTERIEL

La réserve alcaline,
Le sodium,
Le chlore,
Le potassium,
L'électrophorèse des protéines

Sur chaque graphique, la moyenne calculée au cours de ces 30 circulations extra-corporelles totales est inscrite avant et après circulations ainsi que les chiffres maximum et minimum obtenus au cours de ces circulations.

CONCLUSION

Le résultat des 30 premières circulations extra-corporelles dans le laboratoire est exposé.

Ces 30 premières circulations extra-corporelles ont permis de mettre au point, dans un département nouveau, cette nouvelle discipline. Les résultats obtenus nous ont autorisés à transporter cette chirurgie spécialisée sur le plan clinique. Le cœur-poumon artificiel utilisé a été la pompe sigma-moteur avec l'oxygénateur à bulles (Dewall-Lillehei).

Les résultats des examens de laboratoires montrent des variations minimes avant et après circulations extra-corporelles. L'hemoglobine plasmatique a augmenté en moyenne seulement de 18.3 mg. pour 100 c.c. Les globules blancs ont été les plus touchés. Une baisse en moyenne de 2190 a été enregistrée. Les autres éléments de la formule sanguine ont montré des changements insignifiants. Les électrolytes

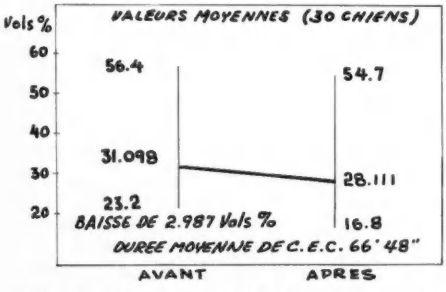


Fig. 17.—Le CO_2 artériel était au début de la circulation extra-corporelle de 31.098 vols en moyenne avec maximum de 56.4 vols et minimum de 23.2 vols et à la fin de la circulation de 28.111 vols en moyenne avec maximum de 54.7 vols et minimum de 16.8 vols donc une baisse de 2.987 vols %.

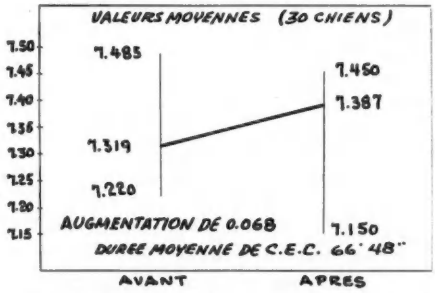


Fig. 19.—Le pH artériel était au début de la circulation extra-corporelle de 7.319 en moyenne avec maximum de 7.485 et minimum de 7.220 et à la fin de la circulation de 7.387 en moyenne avec maximum de 7.450 et minimum de 7.150 donc une augmentation moyenne de 0.068.

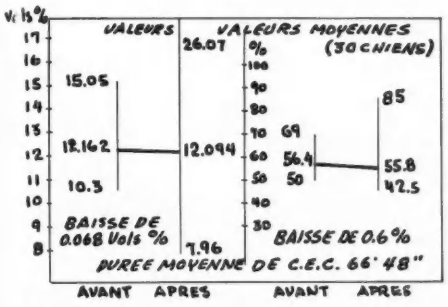


Fig. 20.—L'oxygène veineux était au début de la circulation extra-corporelle de 12.162 vols ou 56.4% en moyenne avec maximum de 15.05 vols ou 69% et minimum 10.3 vols ou 50% et à la fin de la circulation de 12.994 vols ou 55.8% en moyenne avec maximum de 26.07 vols ou 85% et minimum 7.96 vols ou 42.5% donc une baisse moyenne de 0.6%.

ont également été peu influencés. L'étude des gaz a été pleinement satisfaisante. L'oxygène artériel n'a jamais été inférieur à 87% et n'a pas dépassé 103% avec moyenne de 95%. L'oxygène veineux s'est également maintenu en moyenne autour de 55%. Le pH a très peu varié. Ces chiffres indiquent qu'il y a eu un débit sanguin au cours de ces circulations se rapprochant du débit physiologique.

La durée moyenne de chaque circulation extra-corporelle a été de 66 minutes et 48 secondes. Différentes opérations ont été pratiquées au cours de ces circulations. Les différents tableaux résument les résultats obtenus au cours de ces circulations. Nous avons omis volontairement de donner le

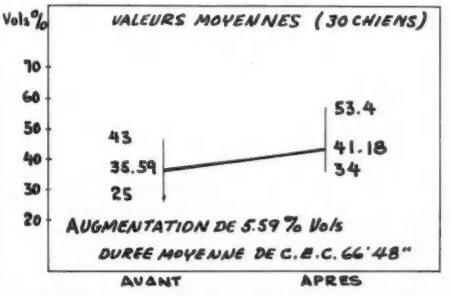


Fig. 21.—Le CO_2 veineux était au début de la circulation extra-corporelle de 35.59 vols % en moyenne avec maximum de 43 et minimum de 25 et à la fin de la circulation de 41.18 vols % en moyenne avec maximum de 53.4 et minimum de 34 donc une augmentation moyenne de 5.59 vols %.

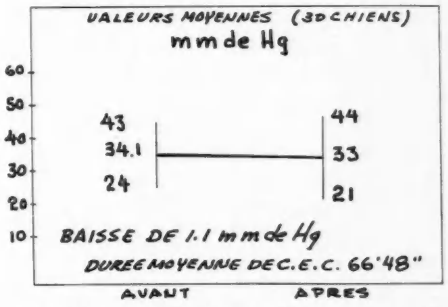


Fig. 22.—Le PCO_2 veineux était avant circulation extra-corporelle de 34.1 mm. de Hg en moyenne avec maximum de 43 et mimimum de 24 et après circulation de 33 mm. de Hg en moyenne avec maximum de 44 et minimum de 21 donc une baisse moyenne de 1.1 mm. de Hg.

pourcentage de survie. Le tableau II résume les causes de la mort qui est survenue soit en fin d'intervention (trois chiens) soit dans les 24 heures (trois chiens) soit de trois jours à 119 jours (12 chiens). Les causes de ces morts sont attribuées soit à l'infection ou à une insuffisance cardiaque. D'autres animaux ont été sacrifiés pour étude anatomo-pathologique. Actuellement 12 chiens survivent et sont en parfaite santé. Parmi ces survivants nous reconnaissons des chiens ayant subi des circulations très prolongées, en particulier le chien N° 64 qui a été 101 minutes et 15 secondes sur l'appareil cœur-poumon artificiel, et a subi une ventriculotomie droite, plus un "patch" sur l'infundibulum du ventricule droit et une cannulation de l'oreillette gauche.

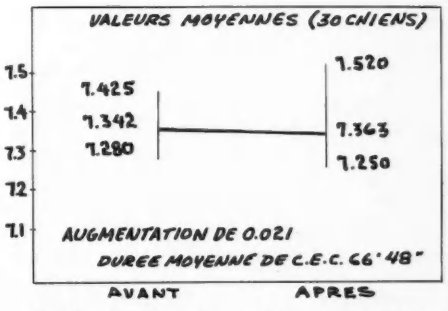


Fig. 23.—Le pH veineux était avant circulation extra-corporelle de 7.342 en moyenne avec maximum de 7.425 et minimum de 7.280 et après circulation de 7.363 en moyenne avec maximum de 7.520 et minimum de 7.250 donc une augmentation moyenne de 0.021.

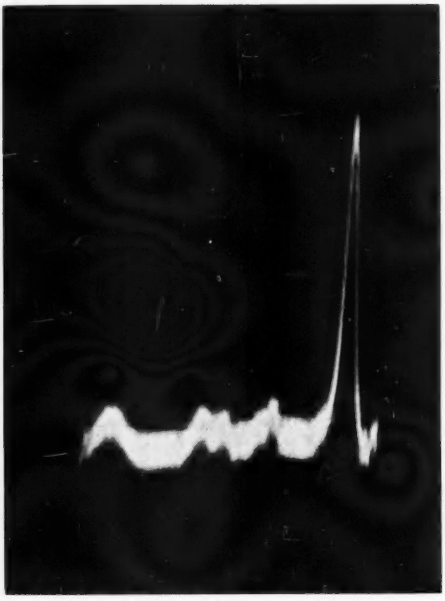


Fig. 24.—Electrophorèse des protéines:
Les protéines totales varient entre 4.68 g. % et 6.50 g. %. La fraction albumine est unique, et représente environ 40% de toutes les protéines sériques. Comme le signalent la plupart des auteurs, il est habituel de reconnaître la présence de 2 à 3 fractions globulaires alpha, 2 à 3 fractions beta et 1 ou 2 fractions gamma. La formule protéinique des chiens dépend de plusieurs facteurs, notamment de l'âge et du régime alimentaire plus ou moins équilibré des animaux.

Sur ces 30 circulations extra-corporelles il n'y a pas eu d'électrophorèse après circulation extra-corporelle pour des raisons qui sont actuellement réglées.

Remerciements

Nous remercions de façon spéciale l'équipe de nos collaborateurs immédiats: Mademoiselle Charlotte Lavoie, i.l., en charge du service interne chirurgical de notre département, et ses collaboratrices: Mademoiselle Jeanne Viel, i.l., Mademoiselle Jacqueline Gilbert, i.l., Mademoiselle Solange Favreau, i.l., Monsieur Marcel Gagné, diplômé en technologie électronique et spécialisé dans le fonctionnement du cœur artificiel, Monsieur Marc Buteau, assistant Technicien et Monsieur Cormier, en charge du chenil. Les photos illustrant le texte ont été prises par le Docteur Anastasiou, Résident en chirurgie à l'Hôtel-Dieu St-Vallier.

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Summary

Statistical results of our first 30 extra-corporeal circulations in the experimental laboratory are presented.

These results stimulated us in attempting open-heart surgery on humans.

The special artificial heart-lung pump used was the sigma-motor type with bubble oxygenator (Dewall-Lillehei).

The blood tests performed before and after extra-corporeal circulation showed variations of no significance.

The plasmatic hæmoglobin averaged 18 mg. % of increase. The white cell counts however dropped to an average of 2190. The other features of the blood picture showed no significant changes. The serum electrolytes were little influenced by the circulation. The gas evaluation before and after circulation was satisfactory. The arterial oxygen never dropped below 87% and was never above 103%, the average being around 95%.

The veinous oxygen has always been maintained around 58%. The blood pH showed little variation.

This data proves that the blood flow during the circulations was close to the physiological point.

Our extra-corporeal circulations lasted 66 minutes 48 seconds on average. Various surgical operations were performed in the course of the circulations. The figures summarize results obtained in those circulations. We have purposely not given any figures on the survival of the animals.

Table II shows the causes of death at various stages of our experimental surgery, i.e., three dogs died at the endpoint of the operation, three within 24 hours and 12 from three to 119 days after operation.

Death of those dogs was imputed either to infection or heart failure. Some animals were killed intentionally for detailed pathological study.

Twelve of our dogs operated upon are still alive and in good condition. Of these, some have had prolonged extra-corporeal circulation: dog No. 64, for instance, had a circulation of 101 minutes 15 seconds during which we performed a right ventriculotomy and a patch on the infundibulum of the right ventricule along with a canulation of the left auricle.

REPAIR OF THE ANTERIOR CRUCIATE LIGAMENT WITH 8 mm. TUBE TEFLON IN DOGS*

M. A. EMERY, M.D., C.M. and O. ROSTRUP, M.D., F.R.C.S.[C], F.A.C.S., *Edmonton, Alta.*

REPAIR OF THE chronic rupture of the anterior cruciate ligament has long been an unsatisfactory procedure from the standpoint of results. Usually these are highly uncertain. Different materials used have been fascia lata, semitendinosis tendon and cutis.[1-3]

Teflon fabric (poly-tetra-fluoro-ethylene) is an inert slippery woven material that produces minimal tissue reaction.[5] It has been used in various procedures such as blood vessel prostheses,[7] repair of hernial defects,[8] and more recently in the repair of neglected torn ligaments.[4] This last experiment was performed on dogs with replacement of the medial collateral ligament of the knee joint.[4] Good results with regard to stability and tissue reaction were obtained. For this reason it was decided to try this material inside the knee joint with the replacement of the anterior cruciate ligament.

MATERIALS AND METHODS

A total of 11 adult mongrel dogs were used in the experiment. In two dogs the anterior cruciate ligament alone was removed to study the effects of this single injury in an otherwise normal knee. The remaining nine dogs had the left anterior cruciate ligament removed then replaced by a strip of 8 mm. tube teflon. The breaking point of the teflon had previously been determined at 115 lb. The opposite knee was used as a control.

Under general anæsthesia of intravenous nembutal, and tourniquet control, a curved medial parapatellar incision was made. The capsule and synovium were opened along the same line. The patella was then dislocated laterally. With the knee fully flexed an excellent exposure of the internal structure of the knee was obtained. The anterior cruciate ligament was then excised. Following this it was noted that there was only moderate antero-posterior joint laxity with the knee in a flexed position. This knee laxity increased when the tourniquet was removed.

The first drill hole was made running from 0.5 cm. to 1 cm. medial to the tibial tubercle to the tibial insertion of the anterior cruciate ligament. A second drill hole was made from the posterior aspect of the lateral epicondyle of the femur to as far distal on the inner joint surface as possible. In all cases the drill hole was not as far posterior as the femoral attachment of the anterior cruciate ligament.

The teflon was then drawn through the drill holes from lateral to medial aspect. The lateral end of the teflon was knotted so that the knot would not pass through the drill hole. The knot was then sutured with 00 silk to prevent the undoing of the knot. The teflon at the medial side of the tibial condyle was stapled to bone and the teflon sutured to itself with 00 silk. This was done with the knee in a fully extended position and the teflon drawn as tightly as possible. The wound was closed. Following this procedure the antero-posterior movement of the flexed joint was reduced compared with that following section of the anterior cruciate ligament but movement was still slightly greater than that of the normal side.

The leg was then immobilized in a single hip spica for three weeks. Nine of the dogs were sacrificed at 12 weeks. These included the two dogs with the anterior cruciate ligament only removed. The remaining two dogs were examined under general anæsthesia for antero-posterior knee joint laxity, then left for an additional three months. Before sacrifice the dogs were examined under general anæsthesia for anterio-posterior joint laxity. Both hind limbs were amputated through the proximal portion of the femur and the foot was removed at the distal third of the tibia and fibula. The joint was opened and the amount and type of joint fluid was noted. All muscle was excised from the specimen and all the femoral-tibial attachments were divided except the anterior cruciate liga-

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This work was supported by Grant 825 from the National Research Council of Canada.

TABLE I.—Antero-Posterior Knee Joint Mobility
Dogs No. 1 to 9—Three Months Duration
Dogs No. 10 and 11—Six Months Duration

Dog No.	*Left knee*	*Right knee (control)*
1	++++	+*
2	++++	+
3	++++	+
4	++++	+
5	++	+
6	++	+
7	++	+
8	++	+
9	++	+
10	++	+
11	++	+

*+ = Normal movement.
++ = Slightly increased over normal.
++++ = Marked increase in movement.

ment on the normal side and the teflon replacement of the other side. Ligament strength was tested with a Baldwin Hydraulic Tester No. 6-35.

Results

1. *Gait.* The two dogs with simple removal of the anterior cruciate ligament had a normal gait on walking but tended to favour the affected leg when running or jumping. The nine dogs with teflon replacement limped for a full month following cast removal but at the end of three months only No. 3 dog limped. The rest had no limp on walking nor any apparent disability on more active movements. The two dogs left at six months had no apparent disability.



Fig. 1.—Articular cartilage degeneration after three month instability.

2. *Range of motion.* At the end of the three weeks of plaster immobilization all the dogs had marked limitation of movement. At the end of three months a slight limitation of flexion was noted particularly when the dog was sitting, but no limp resulted. Dog No. 3 had marked limitation of movement.

3. *Evaluation of antero-posterior knee joint mobility.* The dogs were tested under general anæsthesia with the knee flexed to a right angle.

Dogs No. 1 and 2 had the anterior cruciate only removed.

Dogs Nos. 3 to 11 had the anterior cruciate ligament of the left knee replaced with teflon.

Dogs No. 1 and 2 had marked laxity. Of the dogs with teflon replacement, dogs No. 3 and 4 had marked increase in antero-posterior movement. The rest had a slight increase over that of the normal right side.

4. *Joint reaction. Gross appearance: Three months.* Dog No. 3 had considerable atrophy of the left thigh muscles. The musculature of the other dogs was normal when compared to the other side.

Dogs No. 3 and 4 had a marked increase in synovial fluid. In dog No. 4 this was serosanguineous. In dog No. 3 it was clear in colour. The remainder of the dogs had a slight increase in normal appearing joint fluid.

Dogs 3 and 4 had markedly œdematous and brown stained synovium especially in the region of the patella. In the remainder of the dogs the synovium was less œdematous but still showed gross features of synovial reaction.

The articular cartilage was normal in all dogs except dog No. 3 in which it showed marked degeneration particularly over the femoral condyles (Fig. 1).

The teflon fabric was covered either at both ends (dog No. 3) or in its entirety by white firm tissue which shelled from the teflon with comparative ease (Fig. 2).

In three dogs, Nos. 4, 7 and 8 (Fig. 3) fraying of the teflon was noted (Fig. 4)



Fig. 2.—Gross appearance of three month knee joint with white fibrous tissue covering the teflon.

Indeed in dog No. 4 there was only one strand still intact. In dog No. 3 the tibial insertion of the teflon was more medial than in the other dogs and the teflon was still slightly lax in full extension. The teflon in the other dogs was unchanged.

The femoral and tibial insertions of the teflon were covered with fine white connective tissue.

The teflon appeared to have an erosive effect on bone. A smooth-walled sinus tract was formed which exuded a thick somewhat mucoid brown material when the teflon was removed.

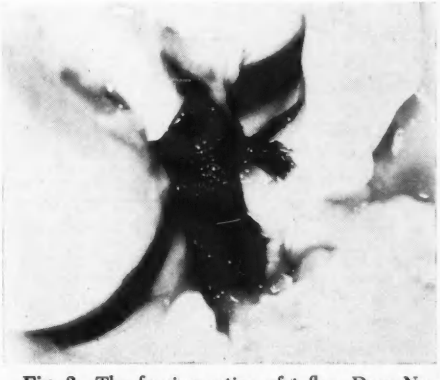


Fig. 3.—The fraying action of teflon. Dogs Nos. 4 and 11 had almost complete fraying.



Fig. 4.—Nearly complete fraying of the teflon.

At six months, dog No. 11 had a slight increase in synovial fluid. The synovium was œdematous. The teflon fabric was

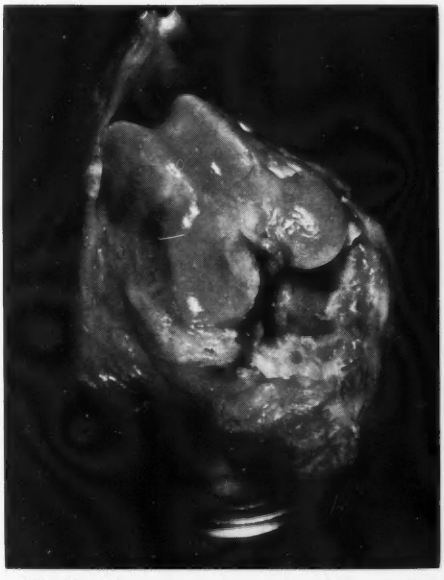


Fig. 5.—A knee joint six months after operation showing almost complete absence of joint reaction.

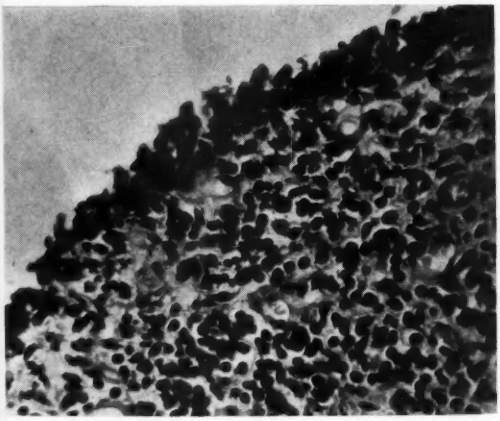


Fig. 6.—Proliferation of the synovial cells and marked chronic inflammatory changes with lymphocytes and plasma cells.

covered with white connective tissue and the teflon fabric partly frayed through.

In dog No. 10 there was no increase in joint fluid. The synovium and articular cartilage looked normal. White connective tissue covered only the ends of the teflon which was intact except for a tiny frayed area in the region of the lateral femoral condyle (Fig. 5).

The erosive effect on bone did not seem any more marked than it was at three months.

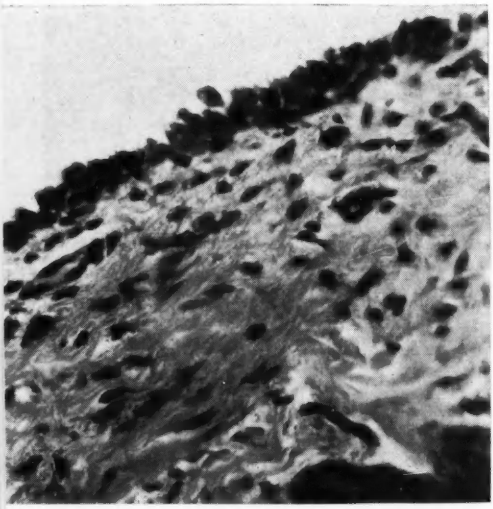


Fig. 7.—Synovium shows little reaction after six months of teflon replacement.

Microscopic appearance.—After three months the synovium showed chronic inflammation with proliferation of the synovial cells and fairly large numbers of lymphocytes and plasma cells. Brown pigment present in macrophages stained for iron (Fig. 6).

After six months the microscopic appearance of the synovium was nearly normal in dog No. 10 (Fig. 7). Dog No. 11 in which the teflon was frayed still showed a chronic synovial reaction similar to that seen at three months. The articular cartilage showed no particular reaction.

The teflon in dogs Nos. 5, 6, 9 and 11 was tested for tensile strength but the chucks of the machine were unable to hold the teflon over 100 lb. This tension was not enough to part the teflon (normal tensile strength 115 lb.). The teflon also held firm at the stapled tibial attachment.

5. *Breaking strength—intact ligament and teflon.*

Table II shows that the normal anterior cruciate ligament was torn free from its tibial attachment in all cases between 70-100 lb. tension. Fraying was noted in dogs Nos. 4, 7, 8, 10 and 11 before testing. Only in dog No. 11 did the frayed area not break before the teflon pulled free from the femoral attachment.

In the remaining dogs the femoral attachment (knotted teflon) pulled through the femoral condyle at 34 lb. to 40 lb. tension.

Discussion

Removal of the anterior cruciate ligament in the dog does not produce much disability although a great increase in the antero-posterior movement of the knee can be demonstrated under general anæsthesia.

TABLE II.—Breaking Point of Ligament (in pounds)

Dog No.	*Left knee (Teflon)*	*Right knee (normal)*
1	—	74
2	—	68
3	—	72
4	Teflon parted	70
5	—	74
6	—	100
7	26	80
8	20	94
9	—	78
10	15	72
11	—	76

Except in dog No. 3 where the teflon insertions were inadequate and in dog No. 4 where the teflon had almost completely frayed through, the teflon gave the joint good stability.

The nearly complete fraying of the teflon in dog No. 4 and the partial fraying in dogs Nos. 6, 7, 10 and 11 are believed due to the constant friction by the articular cartilage and bone on the teflon particularly in the region where it entered the femoral condyle. Aside from this mechanical wear the teflon did not appear abnormal or suffer from any apparent loss of strength. Difficulties in adequately smoothing the inner aspects of the drill holes due to the small joint space no doubt increased the wear on the teflon. This difficulty might be somewhat overcome by working with a larger knee joint. Still, this is a cause for great concern particularly when the teflon must last for years rather than months.

Joint reaction was not great and indeed appeared to subside almost completely in six months. When the teflon remained intact the articular cartilage was not affected.

The type of attachment for the teflon on the lateral femoral condyle was inadequate but the stapled tibial insertion appears to provide an adequate attachment. Therefore it is believed that stapling at both points would provide adequate fixation.

Summary

Eight millimeter tube teflon has been used for replacement of the anterior cruciate ligament in dogs. The dogs recovered nearly full active movement with a normal gait.

Joint reaction was moderate at three months and nearly absent at six months where the teflon remained intact. Stapling and suturing the teflon to itself would appear to provide adequate fixation.

Mechanical wear and tear of the teflon over prolonged periods of time would seem to be the main deterrent in its use. The use of a strip of fascia lata around the teflon could prove of value in the protection of the teflon from mechanical wear and tear.

Acknowledgment

Technical assistance of Mr. W. R. Browne of the Civil Engineering Department, University of Alberta, for his help with the Baldwin Hydraulic Tester, is gratefully acknowledged.

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Résumé

Toutes les méthodes de réparation du ligament croisé antérieur n'ont jusqu'à présent donné que des résultats incertains quels qu'aient été les moyens employés.

Le téflon synthétique est un matériel tissé inerte et glissant qui est déjà employé avantageusement comme prothèse vasculaire, comme complément dans certains déficiences herniaires, et plus récemment dans la réparation ligamentaire, en l'occurence chez les chiens pour la substitution du ligament latéral interne du genou, avec de bons résultats. C'est cette dernière expérience qui a déterminé cette présente étude.

Des 11 chiens dont on s'est servi, deux subirent l'exérèse de leur ligament simplement, alors que les neuf autres eurent en plus la substitution d'un tube de teflon, le membre étant ensuite immobilisé par un plâtre spica simple pendant trois semaines.

La simple exérèse du ligament croisé antérieur chez le chien ne produit que peu d'incapacité, si ce n'est qu'un plus grand mouvement antéro-postérieur du genou peut-être obtenu sous anesthésie générale. Excepté chez les chiens N° 3 et 4 où l'insertion de téflon était inadéquate, les chiens recouvrirent à peu près le plein mouvement actif avec leur allure normale.

Dans le cas où le téflon demeura intact, la réaction articulaire fut modérée après trois mois et à peu près absente après six mois.

Le "stapling" associé à la suture du téflon sur lui-même semblerait assurer une solidité adequate.

L'usure et la déchirure du téflon après un certain temps semblerait constituer le principal obstacle. L'emploi d'une bande de fascia lata protectrice pourrait peut-être solutionner le problème.

SPECIAL COMMUNICATIONS

THE STAGING OF BREAST CANCER
TRIAL OF THE TNM METHOD

A. H. SELLERS, M.D., D.P.H.* *Toronto, Ont.*

In order to facilitate the comparison of results obtained in treatment of cancer of the breast between different centres, not only in our own country, but throughout the world, standardization in the description of the lesion is essential. Professor Sellers is hereby introducing the TNM method of staging beast cancer. Adequate trial of this method, if successful, may lead to considerable progress in this field of surgery by helping to establish a set of uniformly recorded data and universally acceptable statistics.—EDITOR

THE INTERNATIONAL Union against Cancer, through its Committee on Clinical Stage Classification and Applied Statistics,† has recommended a five year clinical trial of the TNM method of staging breast cancer.[1] The trial is international in scope, and began in January 1960. Full details of the method and the trial are available in a booklet entitled "Malignant Tumours of the Breast, Clinical Stage Classification and Presentation of Results".‡

The TNM system provides for the precise description, identification and coding of three components in the clinical extent of disease: the primary tumour (T), the regional lymph nodes (N), and distant metastases (M). Categories are determined at examination before any treatment and remain unchanged, although surgical findings may of course be considered separately. Once the objective clinical findings have been recorded by the examiner, the TNM categories may be examined separately or grouped into clinical stages comparable with those assigned elsewhere by the same system.

To simplify the trial procedure, the Ontario Cancer Treatment and Research Foundation has developed a special form, "Clinical Staging Trial—Breast" (Form CST-B). The form is arranged so that all necessary data for staging by the TNM method may be checked off by the examining clinician without constant reference to any other code or list of definitions. The TNM category and stage may then be assigned by the medical records officer or any other responsible person.

The first version of the form CST-B has been in use in Ontario Cancer Clinics since January 1960. A later version was incorporated in the National Cancer Institute of Canada form and widely circulated in other provinces.

Copies of the form CST-B and instructions for its use may be obtained on application to The Ontario Cancer Treatment and Research Foundation, 69 Bloor Street East, Toronto. Supplies of the form are available to hospitals in Ontario or individual practitioners through the Foundation or any Ontario Cancer Clinic. There is no charge.

*Director, Division of Medical Statistics, Department of Health, Government of Ontario.

†Between 1950 and 1959, the members of the Committee have been: Baclesse, F., Paris; Barajas-Vallejo, E., Mexico, D. F.; Bucalossi, F., Milano; Copeland, M. M., Washington, D.C.; Denoix, P. F., (Chairman), 1954, Paris; Fischer, A. W., Kiel; Hamperl, H., Bonn; Harmer, M., London; Hultberg, S., Stockholm; Lima-Basto, E., Lisbon; Logan, W. P. D., London; McWhirter, R., San Francisco; Racov, A. I., Leningrad; Roxo-Nobre, M.O., (Secretary), Sao-Paulo; Sellers, A. H., (Secretary), Toronto.

‡Copies of the booklet in French or in English may be obtained from Dr. A. H. Sellers, Director, Division of Medical Statistics, Ontario Department of Health, Toronto, Ontario.

REFERENCE

1. Special article, Clinical stage classification of malignant tumours of the breast, *Canad. M. A. J.*, **82**: 319, 1960.

RÉSUMÉ

L'Union Internationale contre le Cancer, par son "Committee on Clinical Stage Classification and Applied Statistics", a recommandé un essai clinique de cinq ans de la méthode TNM pour classifier le cancer du sein. Cet essai a une portée internationale et a commencé en janvier 1960. Les détails sont exposés dans une brochure intitulée "Malignant Tumours of the Breast, Clinical Stage Classification and Presentation of Results".

Le système TNM prévoit la description précise, l'identification et la codification des trois

composants de l'extension clinique de la maladie: la tumeur primitive (T), les ganglions lymphatiques régionaux (N), et les métastases à distance (M).

Les copies de la brochure en français peuvent être obtenues par l'entremise du Dr. A. H. Sellers, directeur, Division des statistiques médicales, Ontario Dept. of Health.

THE NEW ANATOMICAL NOMENCLATURE AS IT AFFECTS SURGEONS*

J. V. BASMAJIAN, M.D.,† *Kingston, Ont.*

Most surgeons have been vaguely aware of the recent official changes that have taken place in anatomical nomenclature, but, understandably, many of them have avoided too close a scrutiny. However, anatomists themselves have shown an amazing agreement on the new terminology—even Soviet anatomists having concurred. Therefore, I feel justified in presenting at this time a review of the features of greatest interest to surgeons. Naturally, the reader interested in details must consult the official list of more than 5000 terms.

The new Nomina Anatomica[1] came into being in 1955 at the Sixth International Anatomical Congress held in Paris. Consequently it has been referred to as the N.A. (Paris). During the Seventh Congress held in New York, the International Committee charged with the duty of correcting the list presented a surprisingly short list of revisions which were approved immediately. It was also agreed unanimously that further changes must be avoided. Thus, at last, we would seem to have a reasonably final official list of gross anatomical terms which in a short time will replace all others in textbooks. Indeed, even now, authors are scrambling to make the necessary changes for future editions and the appropriate claims in their prefaces.

The New York Revision (1960) of the Nomina Anatomica (Paris)[2] has much to commend it but it also will present some surprises for practising surgeons. I shall not deal with all the details but rather give an outline of the salient features and changes. Those who have used the Birmingham Revision (1933) of the B.N.A. (1895) will have the fewest changes to make in their thinking. On the other hand, French trained surgeons will meet with the greatest number of difficulties in spite of the fact that the new terminology was approved in Paris. U.S. trained and European surgeons raised on the B.N.A. will meet considerable changes but undoubtedly many of the new terms will have at least a familiar ring.

First, let us consider the general changes. The most startling of these are the following:

(a) Complete deletion of all eponyms. Thus future surgeons may expect their younger colleagues to be ignorant of many favourite terms such as Hunter's canal, Poupart's ligament, pouch of Douglas, Morison's pouch, circle of Willis, etc.

(b) Complete deletion of all hyphens (N.Y., 1960). This rule holds regardless of the apposition of vowels, e.g., sacroiliac.

(c) Complete deletion of all diphthongs so dear to our British colleagues who have now officially agreed to the change (N.Y., 1960). Hereafter, œsophagus and tænia become, more correctly, esophagus and tenia. (Fœtus of course, has always been incorrect, the correct Latin never having had the diphthong.)

(d) Transliteration of the Latin terms into the local language is fully approved. Thus, for example, brachial artery for arteria brachia is quite correct.

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General Terms

None of the general terms will confuse the surgeon when he sees them used correctly. However in his own usage he may never be comfortable with terms such as *ventral* and *dorsal, cranial* and *caudal* which are offered as alternatives but are not replacements for *anterior* and *posterior, superior* and *inferior*. In connection with the hand, it should be noted that the thumb (pollex) is digit I, the index is digit II, and so on.

Bones

Almost all the terms will give no difficulty. Specially noteworthy is the exclusive use of *axis* for the second cervical vertebra and the final replacement of "odontoid process" by the simpler *dens*. The "thoracic inlet" is now officially the *superior thoracic aperture* with its appropriate antonym. The "optic foramen" now becomes the *optic canal* (because it has length.).

There are some noteworthy terms in the osteology of the limbs. The scapula now has costal and dorsal surfaces; medial, lateral and superior margins; inferior, lateral and superior angles. The glenoid is now a *cavity* instead of a "fossa". The clavicle has sternal and acromial ends.

The medial and lateral lips of the bicipital groove now become the *crests* of the *lesser and greater tubercles* of the humerus and the "spiral groove" has again undergone change, now being the *sulcus of the radial nerve*.

The carpal bones are the *scaphoid, lunate, triquetrum* (note the spelling), *pisiform, trapezium, trapezoid, capitate* and *hamate*. The phalanges will no longer be numbered but referred to as *proximal, intermediate* (or *middle*) and *distal*.

In the lower limb, the term *third trochanter* is applied to the gluteal tuberosity for the first time in the human being. The tarsal bones have retained their most used B.N.A. designations. The *talus* now has no official alternative name; thus students of the future will look blank when an old-fashioned surgeon refers to the "astragalus". The *calcaneus* has had *os calcis* restored to it as an official alternative. The *navicular bone* is found now only in the tarsus and the *cuneiforms* are designated *medial, intermediate* and *lateral* instead of first, second and third.

Joints

Probably the most significant changes in this section come in the names of ligaments although the new terms are quite familiar to most readers. In the axial skeleton the *alar ligament* is the old familiar (paired) "check ligament" of the axis. The ligaments at the neck of a rib are now the costotransverse (previously known to many as the "ligament of the neck"), and the *superior* and *inferior costotransverse ligaments*.

In the limbs, some well deserving ligaments of the joints have regained their lost title of "collateral". Thus we now have the *ulnar* and *radial collateral ligaments of the elbow*. Meanwhile, the "annular" ligament (around the head of the radius) has lost an *n* becoming *anular* because some scholar proved the former spelling to be "wrong" and therefore disturbing to his sensibility. The medial ligament of the ankle is now officially recognized to have the alternative name of *deltoid* ligament because of its shape. Since it is never round, the *ligament of the head* of the femur is no longer the "round ligament". Many will be pleased to learn that, finally, *midcarpal joint* has become official for the line of separation of the two rows of carpal bones, but, inexplicably, the equivalent joint in the foot is the *transverse tarsal joint*. *Subtalar* firmly replaces "subastragaloid", which name is no longer mentioned.

Readers who have been mystified by the variety of ligaments on the dorsum of the sacroiliac joint will be relieved to learn that they have been abolished or, at least, consolidated into one name, the *dorsal sacroiliac ligaments*. Many of us will mourn the passing of the term "semilunar cartilages" of the knee which no longer appears even as an alternative to the slicker and now widely used *menisci*.

Muscles

Most of the familiar terms remain for the muscles. There are some changes of consequence for the intrinsic muscles of

the back but most surgeons can hardly be expected to remember any of them in any case. Most important of these changes is the abolition of the term "sacrospinalis" in favour of the old-fashioned *erector spinae*. The surgeon should be warned, however, that the meaning of this term is limited and it does not include all the intrinsic muscles. The word "lumbodorsal" is replaced by *thoracolumbar* for the well known fascia that covers these muscles.

The most significant change in the terminology of the abdominal muscles concerns the inguinal region. *Conjoint tendon* is demoted to being only an official alternative for *falx inguinalis*, but surely it will not give up without a stubborn fight. The inguinal ligament is recognized as having the following subdivisions: *lacunar* ligament, *pectineal* ligament, and *reflex* ligament.

The most striking changes in name in the muscles of the limbs are the abolition of the well-known term "sublimis" in favour of *superficialis* for the flexor of the fingers and the introduction of *fibularis* as an official alternative to *peroneus* for the three muscles of this name in the leg.

"Hunter's subsartorial canal" becomes the *adductor canal*, the "fossa ovalis" of the fascia lata becomes the *saphenous opening* (*hiatus*). Offered as official alternatives for the *tendo calcaneus* and *quadratus plantae muscle* are *tendo Achillis* and *flexor accessorius*. The committee explained that the use of the name of Achilles was not to be construed as eponymous.

Digestive System

The adoption of the following terms are noteworthy: *cardiac ostium* (of the stomach), *sigmoid* (instead of "pelvic") *colon, rectal ampulla, anal columns, sinuses* and *valves*, and *hemorrhoidal zone* (the part of the anal canal which contains the important venous plexus).

The "lesser sac" of peritoneum no longer appears, being replaced by *omental bursa*, and its "mouth" becomes the *epiploic foramen*.

Respiratory System

The most important changes involve the naming of the tertiary bronchi and the bronchopulmonary segments. It is to be hoped that surgeons will accept the following official terms: *Right Lung*: apical, posterior and anterior segments of the upper lobe; lateral and medial segments of the middle lobe; the apical (or superior), the variable subapical (or subsuperior), medial basal (or cardiac), anterior basal, lateral basal, posterior basal segments of the lower lobe. *Left Lung*: apicoposterior, anterior, superior lingular and inferior lingular segments of the upper lobe; the apical (superior), the variable subapical (subsuperior) and medial basal (cardiac), anterior basal, lateral basal and posterior basal segments of the lower lobe.

Urogenital System

Changes here are generally minor or of interest only to specialists. "Vas" has finally been completely ignored in favour of *ductus deferens*. The urethra is now officially subdivided into *prostatic, membranous* and *spongy* parts. *Perineal membrane* is retained only as an alternative for the *inferior fascia of the urogenital diaphragm*.

Angiology (Including the Heart)

Changes in nomenclature of the heart of greatest significance involve the valves and cusps. *Tricuspid* and *mitral* are reduced to alternates for *right* and *left atrioventricular*. The former has *anterior, posterior* and *septal cusps;* the latter, *anterior* and *posterior*. The aortic and pulmonary valves each have three *semilunar valvules* (not cusps), those for the aortic being *posterior, right* and *left;* those for the pulmonary being *anterior, right* and *left*.

The outstanding change of name for arteries and veins, guaranteed to cause trouble for several decades, is that for the innominates which are now the *brachiocephalic*. There has been a strange enthusiasm amongst anatomists for this change which to some of us appeared to be frivolous. The "internal mammary" becomes the *internal thoracic* for equally pedantic reasons. A new and authoritative list of secondary and tertiary branches of the pulmonary veins was added in the 1960 revision and will be of great interest only to thoracic surgeons.

Nervous System

Most of the new terms in neuroanatomy hold little interest for the general surgeon. Those that do, have been in current usage in English speaking countries. Of course the most striking innovation is the abolition of eponyms already referred to. There are no "Sylvian fissures", "Rolandic fissures", "Clarke's columns" and "aqueduct of Sylvius". They are now (as, of course, they have been for many years) the *lateral* and *central sulci, nucleus dorsalis* and *cerebral aqueduct*. It must be admitted that neither hardship nor great improvement results from these changes. The only advantage is that of regularizing the nomenclature.

The most noteworthy innovation in the peripheral nervous system applies to the name of the eighth cranial nerve which changed its name first in Paris (1955) and again in New York (1960). It is now the *vestibulocochlear nerve*. One is justified in wondering how many years it will take for this name to trip easily off the tongue of the otolaryngologist. The names of the two terminal divisions of the sciatic nerve, after wavering back and forth for almost a century, will now be the *tibial* and *common peroneal* (*fibular*) *nerves*. The latter has *deep* and *superficial* branches (known to some of us previously as the "anterior tibial" and "musculocutaneous" branches of the "lateral popliteal nerve").

In broad outlines, then, these are the changes which appear to me to affect the general surgeon most. I have selected them from a long list of names and undoubtedly have missed words of special interest to individual surgeons. What must be stressed to surgeons is that all anatomists have agreed on an international terminology, something that had never happened before. Officially, there will no longer be several competing systems of nomenclature and so, inevitably, this one system will prevail. Because the International Nomenclature Committee is so determined that no serious tampering will be allowed with the Nomina Anatomica in the future, we can for the first time safely concentrate on teaching one name for each structure, something that has been quite impossible in the past. To expect a sudden switch-over by surgeons trained in the older terms is another matter. The change in the clinical literature will be gradual at first, but surgeons must be aware that, coming as it is from the very roots, this change is inevitable and cannot be held back.

References

1. Nomina Anatomica, Revised by the International Anatomical Nomenclature Committee, Williams & Wilkins Co., Baltimore, 1956.
2. To be published.

Résumé

La plupart des chirurgiens sont vaguement au courant des changements apportés à la nomenclature anatomique, quoiqu'ils n'y aient guère pris d'intérêt. Les anatomistes cependant s'entendent au sujet de la nouvelle terminologie, les Soviets inclus. C'est pourquoi il me semble opportun de présenter les points saillants. Le lecteur intéressé poura consulter avantageusement la liste officielle qui comprend plus de 5000 termes.

Le nouveau "Nomica Anatomica" naquit en 1955 lors du VI congrès international d'anatomie tenu à Paris, et on y réfère comme le N.A. (Paris). Lors du VII congrès tenu à New York, le comité international en charge des corrections ne présenta qu'une liste très succincte de revisions qui furent approuvées sur-le-champ, avec l'acquiescement d'éviter tout nouveau changement. Il semble bien qu'on soit sur le point d'obtenir une terminologie anatomique définitive.

Considérons d'abord les changements d'ordre général:

(a) L'annulation des éponymes, vg.canal de Hunter, ligament de Poupart, etc.
(b) L'annulation des traits-d'union, vg.sacroiliaque.
(c) L'annulation des diphtongues, vg. œsophage et tænia deviennent ésophage et ténia.
(d) La transcription de termes latins est tout-à-fait acceptée, vg. artère brachiale pour arteria brachia.

Termes Generaux

Ceux-ci ne porteront pas à confusion. Cependant, il y aura peutêtre quelque malaise à employer ventral et dorsal, crânial et caudal, qui sont offerts alternativement, mais non comme substituts, pour antérieur et postérieur, supérieur et inférieur. Et ce qui regarde la main, le pouce devient le 1er doigt, l'index le 2e doigt, etc.

Os

Ici, peu de difficulté, sauf en ce qui regarde l'ostéologie des membres. L'omoplate possède maintenant des faces costale et dorsale, des bords médial, latéral et supérieur, des angles inférieur, externe et supérieur.

Les phalanges deviennent proximale, intermédiaire et distale.

Au membre inférieur, l'astragale devient le talus et le calcanéum redevient l'os calcis, et les cunéiformes deviennent le médial, l'intermédiaire et l'externe.

ARTICULATIONS

Les divers ligaments que l'on trouve à la portion dorsale de l'articulation sacroiliaques sont appelés simplement ligaments sacroiliaques dorsaux. Les cartilages semi-lunaires du genoux deviennent définitivement les ménisques.

MUSCLES

La plupart des termes demeurent. Cependant, le mot dorsolombaire est remplacé par thoracolombaire, désignant le fascia qui recouvre ces muscles.

Le tendon conjoint devient la faux inguinale. Le canal de Hunter devient le canal adducteur et la fosse ovale, l'orifice saphénique. Le tendon d'Achille demeure.

Les changements en ce qui regarde les systèmes digestif, respiratoire et urogénital semblent déjà connus.

Du côté des vaisseaux, l'artère mammaire interne devient la thoracique interne.

Le huitième nerf crânien devient le nerf vestibulocochléaire.

Ce qui doit intéresser le chirurgiens, c'est que tous les anatomistes aient acquiescé à une terminologie internationale, ce qui n'était jamais survenu avant. Parce que l'"International Nomenclature Committee" est si déterminé à ce qu'il n'y ait pas de mésentente avec le "Nomica Anatomica" dans le futur, nous pourrons pour la première fois enseigner un seul nom pour chaque élément, ce qui était impossible antérieurement.

URINARY DIVERSION*

"Broadly considered, the term urinary diversion indicates the changing of the normal course of urinary flow from the kidney to the body exterior by surgical alterations of its proper channels, or by conversion to others, surgically created for the purpose, or both. Technical procedures for its accomplishment may be limited and simple or extensive and complex. The simple limited techniques (for example, urethrotomy, cystotomy, cutaneous ureterostomy and nephrostomy), are in the main familiar because of early development and long usage. The more recently devised diversionary procedures are not, because to a large extent they are still in the category of surgical innovations, and concerning them there is less general knowledge and considerable speculation. As their usage increases, some of them will no doubt be discarded and others accepted and standardized according to particular indications.

"It is the purpose of this writing to examine as comprehensively as possible, certain of the more promising of recently developed techniques for urinary diversion. This is done in the hope that such will prove of value to some in the management of disorders where the procedure may be essential to palliation for cure.

"GENERAL CONSIDERATIONS. It may be generally stated that while the procedures for urinary diversion have changed, the indications for its performance have not. These indications may be largely included under a single indication with a few additional ones. This is actual or impending obstruction of urinary flow through its normal channels. Congenital anomalies, inflammation, metabolic disorders, and neoplasms may all produce obstruction of the various parts of the urinary tract. These causes represent theoretically an almost limitless variety of etiological agents of urinary obstruction, all having the common feature of impeding or occluding urinary flow through the various components of the urinary tract. There are however, other indications for diversion, far less frequently encountered which are not obstructive in character. One of these is in conditions where diversion of urinary flow is advisable to create conditions optimal for healing of lesions incident to trauma or inflammation. The other is for the relief of disorders in which severe pain is produced by urinary flow through the normal channels. Thus, the principal indications for urinary diversion may be included under one, a consideration of any two, or all three of the following: (1) relief of impending or actual obstruction, (2) the promotion of healing, (3) the elimination of pain. . . ."

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*CROWLEY, R. T. AND SWIGART, L. L.: *Am. J. M. Sc.*, **240**: 232, 1960.

A LOOK AT RUSSIAN SURGERY – 1960

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THIS SPRING I received a letter from the Deputy Minister of Health of the U.S.S.R. stating that the All Union Scientific Society of Surgeons of the U.S.S.R. was holding its annual Congress of Surgery in Moscow, on May 23 to 28, 1960. I was asked if I would be a guest of the Society for 10 days and present a report of 15 minutes' duration. The four scientific topics for discussion were outlined. This was a fascinating invitation from the point of view of a university surgeon. Judging from the east-west tension developing at that time, it was apparent that our elected representatives could do with whatever small bit of help might be forthcoming from a personal professional contact.

The invitation was accepted with no knowledge of who else had been invited, until further news was obtained some weeks later, at the meeting of the American Surgical Association. Here it was learned that four American surgeons had received similar invitations; Doctor Waltman Walters, Professor Michael DeBakey, Professor Henry Swan and Professor Ralph Deterling. At this time, Henry Swan and I made arrangements to fly to Moscow together on a Russian Aeroflot jet two days before the Congress started. We also expressed the hope, in our reply, that we would have an opportunity to visit the famous Professor Uglov in Leningrad after the Congress.

The Summit Conference fell through a few days before we were to leave, with rather sinister headlines in the American press. This proved a source of worry to two of the American delegates, but no one altered his plans.

At the time I received this invitation I was in the midst of preparing a motion picture showing the technique and results of open heart surgical repair of mitral insufficiency and aortic insufficiency, with cardiac bypass. Both these operations are new and our results have been very satisfactory thus far. This movie was complete and Russian titles inserted with the co operation of Miss Wishart, and her staf of the Medical Art Department, Universit of Toronto. In a further attempt to indicat our good will and respect for Soviet su geons, Russian language slides were als prepared to illustrate my paper on peripl eral vascular disease.

Still imbued with goodwill, I approached Dean MacFarlane and President Bissell of the University of Toronto for official letters of greeting. They both complied and the Dean demonstrated a desire to improve east-west relations by preparing his letter typed in both Russian and English. It was beautifully and formally mounted in a red cloth folder containing a large 14-inch photograph which is now a classic in Canadian medicine. The picture showed Doctors Banting and Best on the roof of the Medical Building with the first dog whose life was maintained with insulin. Ball point pens, coins and some Eskimo carvings completed my goodwill accessories.

While waiting for Henry Swan and our Aeroflot jet at Brussels, I had a fascinating chat with a fellow passenger who proved to be one of President Eisenhower's shadow cabinet. His observations on the current crisis were interesting and instructive. Flight departure was announced and there was an atmosphere of excitement aboard the Soviet jet liner as we prepared to take off for Moscow. It was presumed that there were several passengers, like ourselves, making their first trip into the Soviet Union.

With the concept of Russian supermen and a super race built up in my mind by our western press, I was astonished to find that this was like any ordinary jet plane In fact it was somewhat noisier and less well pressurized than the Boeing 707. I was a comfortable trip with courteous air hostesses and stewards, some of whom could speak a bit of English. It was even reassuring to find that one of the hostesses bore a close resemblance to Queen Eliza

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both II. We were pleased to be joined on the aircraft by that charming gentleman, René Fontaine of Strasbourg, France, who proved to be the only official French guest at the Congress. We discussed pleasant memories of his visit in 1956 to our Cardiovascular Division in Toronto.

On arrival at the Moscow airport, we were met by a very impressive delegation, headed by the President of the Congress, Professor Kuprianov of Leningrad, and which included Professor Petrov, Director of the Sklifosovsky Emergency Surgical Institute and Professor Osipov, Director of the Institute of Postgraduate Studies, as well as junior members of their surgical staffs. One woman surgeon, who spoke French, was assigned to look after Professor Fontaine, and Doctor Victor Krelov was to be guide and counsel for Doctor Swan and myself for the week. As guests of the Soviet Government, we merely identified our baggage at the airport and later found it at the hotel. There were no customs or immigration formalities whatsoever.

Doctor Krelov took Doctor Swan and me by car to the Hotel Moscow, one of the leading hotels in the city. It is centrally placed, facing a square and within a few hundred yards of the Kremlin, Bolshoi Theatre and Gums department store. The hotel is a large stone building that could be called oldfashioned in design. The lobby has a high ceiling and there are large staircases and small elevators. The appointments were functional with an obvious absence of chrome and colour. There was a minimum of accommodation in the lobby for lounging about. Everyone appeared to be about some business and it was apparently not a tourist hotel. Our bedrooms were comfortable and modest, complete with a small television set.

In keeping with our warm and flattering reception at the airport, we took over our rooms at the Hotel Moscow without signing a register, or complying with the usual formality of surrendering one's passport. A very high standard of cordial hospitality and thoughtfulness was maintained during our entire stay in Russia.

Outline of Activities

Sightseeing and a welcoming banquet for foreign delegates was arranged on the Sunday preceding the Congress, which ran from Monday morning until Saturday evening. With the exception of special inaugural and closing sessions, the small group of foreign delegates usually had the choice of visiting one of the Institutes for a special display of their work, or attending the Congress. I personally visited three Institutes and on the fourth morning gave a special lecture at the Vishnevsky Institute. Sightseeing was arranged around this schedule and most of the evenings were taken up with some official function. It was a very tight and active schedule, although we were not compelled to take part in this programme and if we so desired were able to wander off on our own at any time.

Some of us were invited to spend two days in Leningrad after the meeting.

Official cars and small buses took us everywhere we wished to go, usually in the company of a guide. All the guides but one were young doctors and between them they had experts in French, German and English. The non medical guide was an official translator at the Ministry of Health. There was someone on duty in a small room on the mezzanine floor of the hotel at all times for the convenience of the foreign delegates. A private dining room had also been assigned for the small group of foreign delegates.

In most countries of Europe, a little knowledge of the French and German language helps one along satisfactorily, but it is a unique experience in Russia to be completely shut off from communication with no knowledge of the language. Thus this elaborate guide service was most thoughtful and useful.

The Congress

The meeting was held in a grand old building called the Hall of Columns, situated in the heart of the city of Moscow and just across the street from our hotel. There is a most imposing convention hall inside the building, which must have been a grand ballroom at one time with ceilings about 45 feet high, with 24 marble columns, each four feet across and over 30 feet high. Between each marble column is a huge chandelier, and at the end of the room

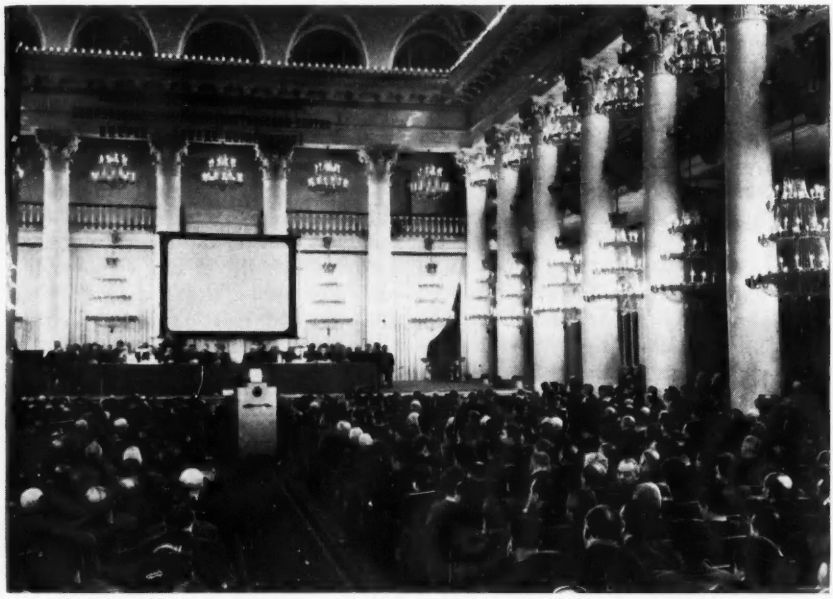


Fig. 1.—The "Hall of Columns" meeting place for the 1500 delegates to the All Union Scientific Society of Surgeons Congress in Moscow. An impressive old hall with marble columns four feet wide, and many elegant chandeliers.

is a large, elevated platform and podium (Fig. 1).

This hall holds 1500 people, so 1500 invitations were sent out to surgeons to attend this Congress. Each of the 17 Russian Republics is sent a number of invitations and they choose their own delegates. It is considered a great privilege to attend and this fact was well demonstrated by the observation that every seat in the hall was filled before the first paper started at 9:30 a.m. and remained filled, apart from a five minute break, until 2 o'clock in the afternoon, which completed the morning session. The afternoon session, from 4 p.m. to 7 p.m., was similarly well attended.

There were two special areas sectioned off on either side of the platform for the foreign guests, so that we were always assured of a seat at any time. By special arrangement, and to accommodate the English speaking guests, they had translation from Russian to English which was received through a small transistor receiver. This was a pocket wireless device complete with ear phones. We could walk out into the corridor and still listen to the English translation. They had a further remarkable show of hospitality by having the entire programme and summary of each paper printed in English. One might expect an arrangement of this kind at an international meeting but it appeared to be a remarkable expression of good will to a small group of about 20 or 30 American and European delegates who could understand English.

The Congress was opened by Professor I. G. Kochergin, Deputy Minister of Health, who outlined the achievements of Soviet medicine during the previous year and spoke at length of one of the fathers of Russian surgery, Professor I. N. Pirogov. This Congress marked the 150th anniversary of his birth.

The business session following the opening speech, was of interest. Professor P. A. Kuprianov of Leningrad, as President of the Congress, read out a list of about 80 names of those officially nominated by a nominating committee to membership in the Administrative Board of the All Union Society of Surgeons. Then in a manner

which made me feel very much at home, he looked out at the audience of 1500 faces and asked if there were any further nominations, and in almost the next breath declared that the nominations were closed. This very august body, leaders of Soviet surgery, were called to the platform.

Professor Kuprianov is a most impressive individual, with a strong, intelligent face and a dignified, well disciplined manner. He appeared resplendent in military uniform, sitting in the centre of the platform, flanked on either side in the front row by Professors Petrovsky, Petrov, Vichnevsky, Kochergin and Bakulev. It was a grand and colourful sight.

There was an interesting system of high intensity spotlights in various places throughout the Congress hall, manipulated by remote control so that the official photographer merely pointed his camera at an object or section of the audience, and it became illuminated.

The formal presentations were indicated on the programme as 10, 15 or 25 minute papers, depending on the importance and seniority of the author. It was interesting that they were held exactly to their time and the meetings ran on schedule throughout the week. On one occasion a speaker was warned by the President that his time was up. He continued speaking and there was an immediate murmur of conversation from the audience. The speaker continued and about one minute later the entire audience commenced clapping in unison. He was forced to conclude at this juncture since no one could hear.

The topics were (1) burns, (2) diseases and injuries of the œsophagus (excluding cancer), (3) conservative and surgical treatment of endarteritis, and (4) tumours of bone. There was one day for each topic with Wednesday and Saturday devoted to business matters. The morning session from 9:30 a.m. to 2 p.m. was assigned to formal presentations on the subject, with the 4 p.m. to 7 p.m. session devoted to discussion. At the end of each day the Chairman of that particular section summarized what he thought were the pertinent points of the day's papers and discussion.

It was interesting that during a very comprehensive and key presentation by Professor B. A. Petrov, outlining a vast experience with surgery of the œsophagus, he gave due credit to Doctor Ross Robertson of Vancouver for his introduction of the use of the jejunal graft.

The foreign guests could choose the particular topic they desired. Doctor Walters spoke on the œsophagus and the other three American delegates and I took part in the programme on peripheral vascular disease. The two British representatives, Professor Charles Rob of London, and Professor Arthur Mackey of Glasgow also chose this subject. Our papers were translated into Russian and a Russian surgeon was assigned to each one of us to read the paper.

I had the honour of having a very senior Russian surgeon, Professor Uglov, Director of the First Medical Institute, Leningrad, read my paper. I took this opportunity to present the formal greetings from Dean MacFarlane and after a brief personal greeting, which I delivered in some memorized Russian, Professor Uglov read the Dean's letter to the Congress. Following this I walked across the platform and presented the formally bound letter, with photograph, to the President of the Congress. This gesture of friendship received a very warm ovation from the assembly. My paper dealt with the results of our surgical replacement of arteries assessed three years after operation, a study which was recently directed by Doctor James A. Key of our Cardiovascular Division. Since the grafting of arteries is a new clinical procedure in Russia they were interested in the long term results. I was told that the Russian slides illustrating this talk were first class translations.

It is interesting that I was feeling rather smug about the fact that I had some Russian slides and said nothing about it to the other guests. At breakfast before the papers were due. Professor Mackey admitted that he had some slides in Russian whereupon it transpired that three or four of us sitting at the table were armed with similar slides, each of us thinking we were quite unique. One of our group suspected that the Russian in his slides was not of very high quality. His suspicions were confirmed when he noticed many smiles and a

good deal of snickering when each of his slides was thrown on the screen.

Doctor DeBakey and I had movies and these were shown at a special session in the Academy of Medical Sciences Building.

Medical Institutes and Surgeons

For the benefit of those who may contemplate a similar trip, I am listing below some of the Institutes in Moscow. This is far from a complete list and only represents those with which I came in contact or visited. These are centres for both undergraduate and postgraduate teaching, as well as research. The director is listed with each Institute.

First Medical Institute
Professor Boris Petrovsky

Sklifosovsky Institute
Professor B. A. Petrov

Vishnevsky Institute
Professor A. A. Vishnevsky

Institute for Thoracic Surgery
Professor A. N. Bakulev—Consultant
Professor S. A. Kolesnikov—Director

Institute for Postgraduate Studies
Professor B. A. Osipov

The Scientific Research Institute
for Experimental Surgical Instruments
Professor M. G. Ananiev

My schedule only permitted me to visit three of the Institutes. With the exception of the Institute for Thoracic Surgery, all the buildings were rather old and similar in design to some of the old hospitals in London, England. There were large wards with beds rather closer together than we might have them, but they were clean and efficient. The patients appeared somewhat subdued according to our standards but cheerful enough. The operating rooms were often very large with high ceilings and as many as two or three tiers of sidewall galleries.

I did not see many shiny, new, chrome plated research laboratories but each Institute appeared to have adequate clinical and animal research areas, usually well equipped with electronic and other types of recording devices. The animal laboratories were often in converted space a one sometimes sees in this country.

One very quickly became conscious of a awareness of seniority on the part of th staff. They still operate a bit on what ha been called the "Herr Professor" system i which there is a chief surgeon, and unde him, at a considerably lower level o seniority, anything up to 10 or 15 assistant One would estimate that half of th assistants were women and some ha been assisting for many years. There wa a very obvious, refreshing and stimulatin eagerness on the part of the younger staf for anything that was new, with particula reference to research.

None of our small foreign delegation had an opportunity to visit any of the outlying or provincial hospitals, although I was invited to one non teaching hospital in Moscow. Certainly the Institutes appear to be efficient and well organized. There was a greater emphasis on gadgetry than is evident in Canada. Each of the operating rooms had closed circuit television and the electroencephalogram and electrocardiogram were used for operations that are ordinarily done without this help in Canada. Their sewing machine for suturing vessels is well known and we saw a special metal ring for vascular anastomosis as well as tantalum clips for closing the bronchus.

Visit to Vishnevsky Institute

About 10 visiting surgeons, with three interpreters, arrived by bus at the Institute. We were suitably gowned and observed an operation from a gallery within the operating room. A Blalock type of anastomosis was being carried out by Professor Vishnevsky on a patient with congenital heart disease.

Professor Vishnevsky works very rapidly and uses no gloves because of a skin condition. He had excellent assistants and two efficient scrub nurses. One sat behind him and received used instruments in a sterile basin, the other stood by the instrumen table in the conventional manner. During the course of the procedure, he walked away from the operating table and happened to stand beside me for three or four minutes. He explained, through a nearby interpreter, that he does this periodically



Fig. 2.—Lunch at the Vishnevsky Institute. One end of the long table in the professor's office where about 10 of us enjoyed a cold luncheon with wine and cognac. The gentleman pointing is Professor Vishnevsky, to his left is the author, and next to him Doctor Henry Swan.

to relax his nerves. One of his patients that morning was operated upon under general anæsthetic and the other under local anæsthetic. This latter technique was used extensively in Russia until recently and during the last five years it has been gradually replaced by current methods of general anæsthesia. I gather that a good deal of the trend has been a result of several visits, and the kindly interest, of Professor Sir Robert Macintosh of the Department of Anæsthesia, Oxford University.

Professor Vishnevsky completed an end to side anastomosis using a small vitallium ring with four outward protruding hooks. This produced a non suture, intima to intima approximation similar to the technique of the single Blakemore-Lord tubes. It was remarkable how rapidly he executed the anastomosis with this ring and the results appeared satisfactory. There was a lecture room in the hospital with closed circuit television where the visiting members from the Congress observed the operation.

Professor Vishnevsky is unique in that he has a definite preference for male assistants. Four or five of his chief assistants and one of the anæsthetists sought me out and plied me with questions regarding hypothermia. It was remarkable that they were well acquainted with all the publications that had come out of our Unit in Toronto and they were keen to know what more we were doing, with particular reference to deep hypothermia. It was a most stimulating hour in a room adjoining the operating theatre. Two of them spoke good English. They were consistently overly kind in assigning credit to the visiting surgeons for research contributions.

This was our first experience with lunch at an Institute. About 1 p.m. approximately 10 of the foreign guests were invited to Professor Vishnevsky's private office for lunch. We found later that this was routine procedure and the Professor's office in each Institute appeared to be large enough to accommodate a small luncheon gathering such as this. The table was laid with an assortment of meats, cheeses, salad, caviar, black bread, etc., with cognac and Georgian wine, and some sweet pastries for dessert. The Professor's office was across the hall from the operating room, and as can be seen from the accompanying photograph we did not remove our gowns (Fig. 2). Actually, there was further operating in the afternoon. Toasts were given to mutual understanding and friendship, and better exchange of ideas. Professor Vishnevsky is very friendly,

alert and keen, and is editor of the journal, "Experimental Surgery".

During the morning's discussion, it was brought out that our Cardiovascular Unit had had success in open heart surgery for mitral insufficiency and aortic insufficiency and the motion picture illustrating these procedures was to be shown at the Academy of Medical Sciences. A special lecture was requested at the Vishnevsky Institute two days later which I agreed with pleasure to deliver.

On returning this time, the hospital lecture hall was filled with about 50 or 60 members of the staff and apparently some visitors from other units. The movie was shown complete with Russian titles, and the problem of direct vision valvular surgery and the future of valve replacement was discussed. Doctor Doneky, one of the assistants, acted as interpreter and was most efficient. The discussion, particularly that relating to the future of this type of surgery, was most interesting. It was followed by the inevitable food, wine, coffee and cakes, and gifts of photographs and inlaid boxes to take home. The photograph shown in Fig. 3 was taken in front of the statue of Vishnevsky's father, founder of the Institute.

Visit to the Institute for Thoracic Surgery

This is a new Institute in Moscow and it is housed in new modern hospital buildings. It is apparently the result of Professor Bakulev's efforts to have a centre devoted entirely to thoracic surgery and related research. Professor Bakulev was formerly the Director, but he has recently become a consultant and the current Director is Professor Kolesnikov.

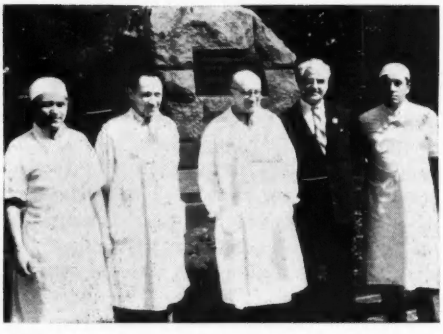


Fig. 3.—Professor Vishnevsky (central figure), Professor Ananiev (dark suit) Director of the Institute for Surgical Instruments, and members of the surgical staff of the Vishnevsky Institute.

Here we were met again with the usual warm reception and observed a mitral valvoplasty from an overhanging, modern viewing gallery in the operating room. This operation was carried out by the standard technique with a lateral incision. During ward rounds, we saw a patient on whom a ventricular septal defect had been closed with cardiac bypass about one week previously. This young lad was in excellent condition and it was ascertained that this was one of their first clinical cardiac bypass operations. Mr. Cleland and Doctor Melrose of England had previously visited the Institute for several weeks and demonstrated the technique. They were using a bubble oxygenator of their own design.

The hospital is divided into five services; one for acquired heart disease, one for congenital heart disease, one for disease of lung, one for disorders of œsophagus, and one for diagnosis and therapy, each of about 50 beds. There is a fine new clinical laboratory and extensive animal laboratory quarters in a converted building adjacent. Their research is what one would expect in a progressive cardiovascular centre in this country. They are dealing with problems relating to extracorporeal circulation, deep hypothermia, plastic valves, plastic bronchus and coronary artery disease. Following this extensive tour, we had a typical delightful lunch once again.

This appeared to be a very progressive Institute which we understood was receiving considerable government support. Professor Bakulev is a most pleasant, intelligent and energetic man who is probably one of the most senior surgeons in Russia today.

Visit to the First Medical Institute

This Institute is directed by Professor Boris Petrovsky and would appear to be not only first class medically, but important professionally and politically. Professor Petrovsky is the very capable editor of the journal Khiirurgie and seemed to be a

rather more serious man than his opposite numbers in the other Institute.

Once again, we encountered an enthusiastic group of assistants, full of questions and keen to improve their technique and learn about current research. Their frank admiration and profound respect for any intellectual accomplishment, whether it be in science or the arts, has been described before and was strikingly illustrated by our association with this institute's surgeons and anæsthetists.

Professor Petrovsky has had success resecting ventricular aneurysms of the heart following cardiac infarction. He operated upon one such case, which proved inoperable, but we saw a movie illustrating the technique. Lunch in the Professor's office with the usual repast and toasts was complete with the giving of presents to all the foreign visitors in the form of books on Soviet art. Fig. 4 shows Doctor Olga Koljutonaja, chief anæsthetist, Professor Boris Petrovsky and Doctor Demetri Venidiktov leaving the First Medical Institute.

Impressions of the Institutes

From what one could gather on a brief visit, it would appear that general surgery in these teaching institutes is done as well as one would see it performed in any first class Canadian or American centre. In peripheral vascular surgery, Russian surgeons are just starting to perform arterial replacement surgery for obstructive disease and aneurysms of the arteries. The most comprehensive clinical report at the Congress was from the First Medical Institute where a total of 28 cases were reported including aneurysms and occlusive disease. They are all doing some of this work experimentally testing our teflon and dacron prostheses as well as one of their own, a material named Levson.

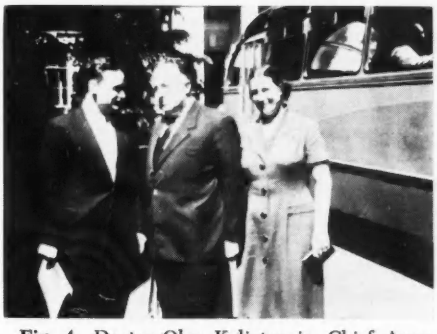


Fig. 4.—Doctor Olga Koljutonaja, Chief Anæsthetist, Professor Boris Petrovsky, and Doctor Demetri Venidiktov leaving the First Medical Institute. Professor Petrovsky is Director of the Institute and editor of the leading journal Khiirurgie.

In heart surgery, closed heart techniques for patent ductus, Blalock procedures and surgery for mitral stenosis are performed by the standard techniques as well as one would see anywhere. In open heart surgery with hypothermia, some of the centres have a limited experience. Cardiac bypass surgery, using the heart-lung pump, is in the experimental stage with the possible exception of that conducted in the Institute for Thoracic Surgery, where a few clinical cases have been operated upon. Thus they are two to five years behind us in experience with peripheral vascular and open heart surgery. There appears to be a good deal of emphasis on surgery for cardiovascular disease in all centres.

One striking feature already mentioned, is their knowledge of American scientific literature. They appear to be quite up to date on all publications and in special fields, such as hypothermia, they quoted articles published many years previously. It was gently and diplomatically suggested to me on one occasion that it was apparent we did not read their literature to the extent that they read ours. That was perfectly correct, not only pertaining to myself but to other members of the party as well. In fact, I found on returning to Toronto that our university library did not have subscriptions for the four leading surgical journals in Russia. This is an obvious oversight (for in at least one journal Khiirurgie there are English summaries at the end of many articles). It is hoped that this will be rectified. As an aid to other libraries the main surgical journals, with the editor and subscription rate per year are listed here. All these journals can be obtained by writing to the one address.

Vestnik Khiirurgie	12 Issues	72 Roubles
Editor Uglov		

Experimental Surgery Editor Vishnevsky	6 Issues	18 Roubles
Khiirurgie Editor Petrovsky	12 Issues	72 Roubles
Chest Surgery Editor Bakulev	6 Issues	36 Roubles

Available through:
Ministerstvo Mezhdunaradnoy Torgovly,
V-O Mezhdunarodnaya Kniga,
Smolenskaya Sennaya Ploschad D 32/34,
Moscow, Russia.

As has been mentioned there was evidence of experimental as well as clinical research going on in all the Institutes visited. The amount of experimental research being directed by clinicians is not as great as one would find in a Canadian or American university but one would gather that the amount is increasing.

One hears a great deal about grafting a second head on a dog. Actually, it is a skillful technical procedure but, as in all organ transplant work, these parts only live a week or two and there is a limit to the value of the operation. Organ transplant, including that involving the heart, and regrafting of a dog's amputated leg is under study in the Sklifosovsky Institute by Doctor Danikov, as it is in several centres in Canada and the United States.

One gathered the impression that any piece of research that did prove successful and unique, such as the sewing machine or the grafting procedures, obtained an incredible amount of publicity abroad. It does not alter my belief that their clinical research programme is not nearly as varied or extensive as we have in North America.

Sightseeing and Social Activities

In typical western tourist fashion I took in as many sights as possible in a superficial and sometimes hurried manner. One of the most striking was a visit to what is called the "Pavilion". This is a huge glorified exhibition ground consisting of about 70 buildings or pavilions covering several square miles. This whole pavilion project would appear to be primarily educational but the buildings and grounds are so unique in design and there is so much of interest that it serves as a form of entertainment. Roughly 40 of these pavilions are assigned to the exhibition of subjects in such fields as agriculture, books, electronics, health, history of the Soviet Union, organization of the Soviet Government, etc. These pavilions are of common interest to everyone. In addition, each of the 17 Republics has one to three pavilions demonstrating life in that area and matters of economic and cultural interest. The most modern plane will be open for inspection and one can sit by fountains and eat ice cream. This is apparently one means of unifying the Soviet Union which is made up of 17 Republics, each with a different language.

The university is a huge building on the outskirts of Moscow, 22 stories high in the centre column, and with an enrolment of 40,000. It houses everything under one roof, including administration, classrooms and residences. There is one large wing of this huge building devoted to living quarters. There are two students assigned to a small functional suite with a communal kitchen at the end of the corridor, with men students occupying one wing of a floor, and girls the other.

Perhaps the most impressive experience was an evening at the Bolshoi Ballet where we saw Swan Lake. The incredible Bolshoi Theatre, combined with faultless dancing and fine music proved an emotional experience for me. The theatre has an extremely high ceiling with six tiers of gold loges. I sat in the front row in the orchestra seats and listened to music played by an orchestra of men in black ties (the only time we saw these in Russia). Besides the superb dancing, the costumes were most dramatic. We did not see Ulanova, but the young lady who took the lead was considered by our Soviet hosts to be almost as good as this prima ballerina.

I saw the interesting Tretijakov Art Gallery named after the man who many years ago decided it was time to sponsor Russian art. Gums store which has been described many times is an extensive glass arcade, and the metro underground railway appeared almost too ornate in some places with its beautiful tiled walls, but

it was clean and efficient. The Kremlin churches perched on high ground are ornate evidence of a bygone religious era.

It was a striking experience to visit the mausoleum containing the tombs of Lenin and Stalin. Each day of the week there is a queue, two or three deep, extending along the Red Square for about a quarter of a mile. On being brought to the head of the queue by the guide there was no objection to my being allowed by the guard to step in front of a group of young Pioneers, 10 years of age, that had been in the line for three and one-half hours. They were still well disciplined and one had the feeling that this was what they wanted to do rather than something they were forced to do. The two bodies are beautifully preserved, strongly floodlit and one passes within about 10 feet of them.

On two late evenings I wandered about the streets of Moscow. The very effective, night time illumination of Red Square, including the mausoleum and the famous and bizarre St. Basil's Cathedral, was an experience I shall long remember. There was an hourly change of the guard in front of the tomb.

A formal welcoming dinner was given by the Administrative Committee of the All Union Society of Surgeons for the official foreign guests. Besides the American, British, and French guests already mentioned, Professor Husfeldt was invited from Copenhagen, two representatives from Sweden, five representatives from China and one or two from each of the Russian controlled countries in Europe. There were English and French speaking doctors and interpreters sprinkled throughout the gathering to permit adequate conversation. Following the initial toast of welcome by the Chairman, representatives of each country in turn gave a toast. China led off, through an interpreter, followed by Doctor Walters for the United States and the toasting went on for about 40 minutes. This was carried out, fortunately, during the initial cold meat and salad course, and all the speakers stressed the desire for friendship and better exchange of ideas. The Chinese delegation appeared very disciplined. They were all dressed the same in a very modest type of uniform and kept rather to themselves throughout the period of the meeting and did not appear to encourage conversation. I thought they were somewhat different from my concept of the average, pleasant, friendly Chinese.

I was particularly struck by the absence of excessive drinking. There was one drink before dinner and a rather modest amount of cognac and wine consumed during dinner. The Russians would appear to have a rule about drinking at official functions.

This dinner was more or less duplicated on Saturday evening at the end of the Congress. Once more, each of the countries gave a toast including expression of their thanks. There was a modest attempt at humour in some cases, but by and large most of them were rather serious and the Soviet members present genuinely appeared to appreciate the oft repeated platitudes. I must say that the American representatives and I were given every consideration during these social gatherings.

I was invited to four private dinners and was able to accept three of these invitations. One was by Professor Uglov of Leningrad. He had a quiet, small party in his hotel suite with Professors Swan, Charles Rob, Ralph Deterling and myself with Doctor Venidiktov as interpreter. Professor Uglov speaks fairly good English. He is a small, wiry, unassuming man, with a ready smile and a very charming manner. He is soft spoken and exudes genuine friendship. It was interesting to see this man, born 1000 kilometers from the nearest railroad in Siberia, and now one of the top surgeons in the Soviet occupying the royal suite of the national hotel with its high ceilings, inlaid furniture and ornate walls and ceilings.

A second small private hotel dinner given by Professors Bakulev and Kolesnikov was very gay in the finest Russian tradition and a certain amount of endurance was required in order to enjoy the whole evening.

Two of the invitations were to private homes but I was only able to accept a dinner with Professor Shabonov, who had visited us in Toronto. He is a big, coarse haired, friendly fellow who is Director of

the non teaching Botkin Hospital in Moscow. As you know, there are no private homes as such in Russia. One has rooms in a large, plain apartment house, and the greater one's skill and the more important one's status in labouring or intellectual circles, the better living quarters one is assigned. Professor Shabonov occupied a six roomed suite in the sixth, or top floor of one of these grey stone apartment buildings that run endlessly in Moscow. There was a tiny elevator that would accommodate three passengers and the apartment, although not luxurious, gave the atmosphere of comfort and culture. All the rooms were small but there were book cases up to the ceiling of the living room and extending down the corridor to the front door. As in the case of many Soviet surgeons, Professor Shabonov's wife is also a doctor and he has a son an engineer and a daughter, age 22, who is studying art. The family of five occupy this apartment.

I was the only guest at this dinner and Doctor Venidiktov came as interpreter. This was the most interesting experience of my visit since it was not only an insight into the home life of a Russian family but towards the end of the dinner we discussed politics and international problems and in the presence of Doctor Venidiktov, who I would gather is an important medical political figure in Moscow. Space does not permit an elaborate description of this party. It was essentially friendly and cheerful with joking and good humour. The 22 year old daughter was a charmer by any standards. She had great fun trying her English. Doctor Shabonov told me, through Venidiktov as interpreter, that his wife was so disturbed at the prospect of having me as a guest for dinner that for one week he and his daughter had to encourage her and they told her daily that she was the finest cook in Moscow in order to maintain her morale. Mrs. Shabonov, who is a senior physician in her own right, took this joke in good spirit.

The meal involved initially a small drink of vodka in the living room, then some cognac with the cold meat, caviar, cheese, and salad course. Following this a huge bowl of soup was presented and it is one of their important food items. The Russians, by and large eat more than we do and the size of the bowl was too much for me. Actually, I was told that in the small visitors' dining room in the hotel, they served guests their soup in children's soup dishes. Following the soup there was a steak of excellent quality and the dessert was a pastry. The dining room was very small and the decor gracious but not elaborate.

Doctor Venidiktov asked me if I would like to discuss international events, a subject that had been avoided throughout our stay. He said, with a smile that "The west is so suspicious of us that we thought that if politics were discussed you would think that was one of the reasons why you had been invited to the Congress." The discussion was brief and it was carried out very pleasantly and calmly with absence of strong opinions. As far as I could gather, they appeared to want to know what motivates the Americans to carry on such an aggressive cold war. I had the feeling that they did not appreciate what a source of trouble international communism has been over the years to Americans and Canadians. It was stressed that the average Russian would like an end to the cold war so that the Soviet Union could divert some of their money and energies to building adequate housing, which is in extremely short supply. Apparently, it is possible for a professional couple to have to live in one room, and one of their strongest desires is to have adequate living facilities with some privacy.

Week End in Leningrad

We left the Saturday banquet a bit early and proceeded to the Red Arrow train which runs from Moscow to Leningrad. On the invitation of Professor Uglov, the party included Swan, Boliga of India, Doctor and Mrs. DeBakey, and Husfeldt of Copenhagen. The compartments were small, plain but comfortable, resembling a British railway carriage. Tea with cake was served by two lady attendants.

After an overnight trip, we were met



Fig. 5.—A relaxing week end at Professor Uglov's country home. From left to right: the author, DeBakey, Husfeldt (Copenhagen), Uglov, Boliga (India) and Swan.

by Professor Uglov's staff at the station on a brilliant sunny day. We were taken to an hotel and later driven 30 kilometers into the country to Professor Uglov's country home, mute evidence of the high position he holds in the Soviet Union. On the way we passed a similar, or perhaps more elaborate country home owned by one of the Soviet's leading poets. Professor Uglov also owns an apartment in the city of Leningrad. We had two enjoyable cold meals, went for a walk and generally had a lazy, pleasant time. The photograph, Fig. 5, shows Professor Uglov and his relaxed guests. Mrs. Uglov played the piano for us and sang extremely well.

We visited the First Medical Institute the following morning, where we saw a lobectomy and mitral commissurotomy performed by Professor Uglov and his excellent assistants (Fig. 6). He has about as elaborate an investigative unit as we had seen and a very fine dedicated staff. Once again the buildings were old. The clinical investigative unit is a converted area but they were carrying out many interesting lines of research.

Professor Uglov showed us some of Leningrad and I visited the renowned Hermitage with its world famous collection of European art. This was where the Czars and Russian nobility lived a great deal of their time and of course not only disregarded Russian art in their collection, but felt compelled, as an index of refinement, to speak French.

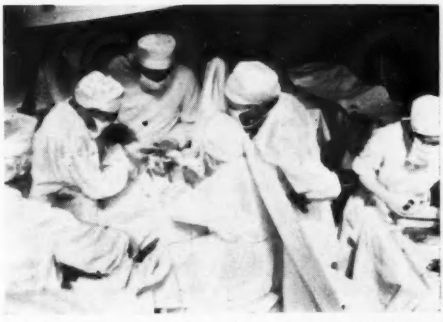


Fig. 6.—A mitral commissurotomy by Professor Uglov of Leningrad. One scrub nurse sits out to one side, immediately behind the operator. There is a sterile basin between them in which used instruments are deposited. There is a second scrub nurse, not shown, in the conventional position at the foot of the table. This was a common arrangement. The two senior assistants on this large staff were women surgeons.

Professor Uglov told us of the difficult time Leningrad had during the war. They were under siege by the Germans for three years, during which time 3000 missiles per day landed on the city, and no building escaped damage. He was in the city during the siege and is a man of fine reputation. It was rather significant that he told us that 100,000 people died of starvation in Leningrad alone during that three year period. Apparently, the bodies were collected each day by a government hearse. It was further evidence of the incredible suffering that Russia experienced during the second great war. Their reported overall death toll is staggering.

We said goodbye to Professor Uglov and his assistants, and were given the usual presents of books and fine Georgian wine. I gave him an Eskimo carving which he appeared to appreciate.

Impressions

At the risk of being stamped as a 10 day authority, I did feel compelled to record some of my impressions. One of my strongest impressions, meeting on a professional level, was that these are ordinary people, less aggressive and perhaps friendlier than some western European nationalities. One does sense a good deal o discipline and official supervision of thei activities which would prove most frustrat ing to us. They are proud of thei achievements, ashamed of their shortcom ings and fearful of war. Young boys, 1 or 15 years of age will have had three year schooling in English. Recognizing America dress they will ask to speak to you to tr out their English. Everywhere in Moscov one sees these groups of young children with distinctive neck scarves, takin supervised tours of art galleries, museums etc. (Fig. 7).

In science they appear to have put a large amount of their energy, resources and brains into two or three projects, such as balistics, sputniks and power dams, and have given their success in these fields an immense amount of publicity. They are content to use copies of western refrigerators and anæsthetic machines. Hearing constantly of their great successes in a few things, one loses sight of the fact that they are a poorer country than we are with regard to general standard of living, transportation, building, hospitals, hotels, dress, etc. After having visited five different offices and taken one and one-half hours to have an air ticket changed, one had the



Fig. 7.—One of the many groups of young "Pioneers" on a cultural tour of the Red Square in Moscow.

feeling that bureaucracy of this kind will never compete in material things with the western free enterprise system. Their method of education and their desire to learn is remarkable, however, and we should continue to study their techniques.

Clinical research is very often monotonous, frustrating and discouraging work. It is the occasional success that gives one the erroneous impression that research is dramatic and exciting. In order to maintain a young man's interest in clinical research, he must be given professional prestige and authority as he progresses in seniority and achievement. The success of Soviet research may depend to a degree upon the ability, with their oldfashioned seniority system, to offer that necessary stimulus.

I felt that the ordinary people in Russia and we in the west are not well enough informed of each others motives and mode of life. On the one hand we have a biased free press, on the other a controlled press. Perhaps the answer lies in better communication. Perhaps the Canadian government could assign the cost of one military plane to pay a group of experts to study and implement means of real communication.

I took a small step by arranging to have Doctor Ronald J. Baird, who is joining our staff at the Toronto General Hospital, spend three months at the Institute for Thoracic Surgery in Moscow. To the best of my knowledge this is the first time that an American or Canadian will have received postgraduate surgical education in Russia. The idea of having Doctor Baird study with them was received warmly. It is hoped that this will be a first step in better future communication at the professional level.

RÉSUMÉ

L'auteur relate ses impressions d'un voyage en Russie pendant lequel il assista à titre de conférencier invité au congrès annuel de la Société scientifique des chirurgiens d'U.R.S.S. tenu à Moscou du 23 au 28 mai dernier. Comme preuve de bonne volonté, l'auteur s'arma de diapositives rédigées en russe afin d'illustrer sa communication, ainsi qu'un message du doyen et un autre du président de l'Université de Toronto. Les visiteurs furent accueillis à l'arrivée par une délégation imposante tirée des cadres des principaux instituts médicaux de Moscou. Une fois réglées les formalités douanières ou autres (qui d'ailleurs avaient été réduites au plus simple minimum), les visiteurs furent logés à l'hôtel Moscou où les attendait une hospitalité qui ne devait pas se démentir de tout leur séjour dans ce pays.

Le congrès comprenait un programme bien rempli, tant de séances scientifiques que d'événements sociaux. Les guides affectés aux médecins étrangers étaient eux-mêmes de jeunes médecins possédant le français, l'allemand ou l'anglais. Les réunions furent tenues dans la salle dite "des colonnes", qui peut contenir 1500 personnes. L'invitation à ce congrès constitue un honneur très recherché au sein de la profession médicale; la présence assidue de tous les délégués des différentes républiques en est la preuve. On avait organisé à l'intention des visiteurs étrangers un système d'interprétation simultanée du russe à l'anglais; les programmes comportaient aussi des résumés en anglais.

Bien que présidé par le Pr Kuprianov, c'est au Pr Kochergin que revint l'honneur d'ouvrir le congrès. La durée des communications était fixée à 10, 15 ou 25 minutes selon l'importance du sujet traité et le président de l'assemblée observa toujours cet horaire de près si bien que toutes les communications purent être présentées et que les séances se terminèrent à l'heure indiquée.

Le congrès comportait quatre grands thèmes: à savoir, les brûlures, les lésions non-néoplasiques de l'œsophage, le traitement conservateur et la chirurgie dans les artérites et les tumeurs des os. Les séances se tenaient de 9:30 à 2:00 et de 4:00 à 7:00 heures. A la fin de la journée, le président de la section donnait un résumé des faits saillants de la séance. Les communications des visiteurs furent traduites en russe et chacune fut lue par un chirurgien que l'on avait désigné pour cette tâche. L'auteur choisit de traiter des résultats lointains obtenus dans le remplacement des artères, tels que revus après un recul de trois ans.

Dans ses rares loisirs, il visita plusieurs instituts de médecine et de chirurgie. Il offre même au lecteur qui serait tenté d'en faire autant, une liste des principaux instituts dont il recommande la visite. Même si l'état des bâtiments et des locaux semble quelque peu vieillot à comparer à celui de la plupart des établissements nord-américains, il ne s'y fait pas moins du bon travail. Le personnel de ces instituts est imbu de la notion de hiérarchie de sorte que le "patron" est le maître incontesté dans chacun d'eux. Environ la moitié des assistants sont des femmes. Tous manifestent un vif intérêt aux progrès dans les différents domaines de la médecine. L'auteur prétend avoir décelé un faible pour l'appareillage mécanique ou électronique qui semble une manifestation d'un culte de la machine.

Les visiteurs étrangers assistèrent (en spectateurs) à une anastomose de Blalock pratiquée par le Pr Vishnesvsky, à l'institut qui porte son nom. Le professeur opère rapidement et sans gants. Ses assistants semblent fort au courant des plus récentes découvertes dans le domaine de l'hypothermie et ils assaillirent l'auteur de questions se rapportant à ses propres recherches. L'auteur avait apporté une pellicule cinématographique illustrant sa technique de correction à cœur ouvert de l'insuffisance mitrale et aortique; on le pria alors de prononcer une conférence pour en accompagner la projection. Cette communication fut suivie avec beaucoup d'intérêt de la part des auditeurs.

Les visiteurs étrangers virent ensuite l'Institut de chirurgie thoracique où ils assistèrent à une valvuloplastie de la mitrale. Cet établissement très moderne comprend cinq services d'environ 50 lits chacun et qui se répartissent comme suit: maladies congénitales du cœur, maladies acquises du cœur, maladies des poumons, maladies de l'œsophage et enfin, diagnostic et thérapie. Cet institut était dirigé jusqu'à récemment par le Pr Bakulev qui était l'hôte pour la circonstance.

Au cours de leur visite au Premier institut médical, dirigé par le Pr Boris Petrovsky, les délégués étrangers furent reçus avec la courtoisie habituelle et l'intérêt soutenu du corps médical russe. L'auteur est d'avis que la chirurgie générale pratiquée dans ces institutions est de qualité comparable à celle que peuvent offrir les meilleurs centres canadiens ou étatsuniens. En plus du Teflon et du Dacron, les Soviétiques possèdent leur propre matériel de prothèse vasculaire qu'ils nomment Levson. S'ils ont su maîtriser la technique opératoire à cœur fermé, ils n'ont encore acquis par contre, dans certains centres, qu'une expérience limitée de la technique à cœur ouvert, sous hypothermie, dont la mise au point est d'ailleurs assez récente. Leurs dérivations cardiaques par pompes cœur-poumon ne sont dans la plupart des cas qu'au stage expérimental. Tous ces chercheurs semblent très bien renseignés des progrès de la science nord-américaine—beaucoup plus que nous ne le sommes de leurs propres réalisations.

Profitant de son séjour, l'auteur visita le Pavillon, sorte de foire permanente contenant des exhibits de toutes les Russies. On lui montra également l'Université de Moscou, gratte-ciel de 22 étages servant à 40,000 étudiants, le théâtre Bolshoi où il assista à une représentation de Lac des cygnes, la galerie d'art Tretijakov, nommée d'après le "découvreur" de l'art autochtone et enfin les dépouilles mortelles de Lénine et de Staline dans leur mausolée.

Aux banquets qui marquèrent l'ouverture et la clôture du congrès, on porta des santés et on échangea des protestations d'amitié pendant près de trois quarts d'heure. Tous ces vœux et ces allocutions étaient prononcés sur un ton solennel rarement teinté d'un faible humour. L'auteur fut invité à quelques réceptions intimes dont une chez le Pr Shabonov qui poussa la courtoisie jusqu'à offrir de discuter la situation internationale et la guerre froide. Il eut aussi le plaisir de passer la fin de semaine près de Léningrad à la maison de campagne du Pr Uglov (à qui on accorde ce luxe en vertu de son poste élevé dans le corps médical). L'auteur garde un souvenir ému des quelques heures passées en sa compagnie.

L'impression qui lui est restée de son voyage chez les Russes est celle d'un peuple amical, moins aggressif qu'on est porté à le croire, soumis à une discipline et à une surveillance officielle que nous pourrions difficilement tolérer. Ces gens sont fiers de leurs réussites, gênés de leurs faiblesses et craintifs de la guerre. Leur niveau de vie n'atteint pas le nôtre et la bureaucratie reigne en maître chez eux. Nous gagnerions autant qu'eux à étendre la connaissance réciproque de nos deux peuples. Comme preuve de sa détermination dans ce sens, l'auteur a recommandé que l'un de ses assistants fasse un stage de trois mois à l'Institut de chirurgie thoracique de Moscou. Ce projet est actuellement à l'étude.

CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year, and starts with the January 1 issue of each year. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

INSTRUCTIONS TO CONTRIBUTORS

Manuscripts

Manuscripts of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text and should be set out in accordance with the *Cumulative Index Medicus* abbreviation of journal name and general style. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. References to books should include in order: author's name; title of book; title of publishing house; city of publication; number of edition (e.g., 2nd ed.); year of publication.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

Language

It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

The Royal College of Physicians and Surgeons of Canadà

NEWSLETTER

Earlier this year, the Trustees of the R. Samuel McLaughlin Foundation announced the creation of a visiting professorship in Canada, the professorship to be known as The McLaughlin Foundation Edward Gallie Visiting Professorship in The Royal College of Physicians and Surgeons of Canada.

In this way, a suitable memorial to the great Canadian surgeon, the late Dr. William Edward Gallie, was established and fittingly dedicated to the free interchange of ideas and teaching methods.

It is the wish of the founders that the visiting professor should visit various medical schools across Canada, taking part in their teaching programmes, lectures, conferences, clinics and rounds. Non university centres will also be included in the itineraries.

The selection of the visiting professor rests with a committee of representatives from The Royal College and the Association of Canadian Medical Colleges, subject to final approval by the Trustees of the Foundation. The responsibility of the Committee includes the choice of areas to be visited and the length of time which the visiting professor will spend at each.

Although the visiting professor may be chosen from any country in the world, and may represent any field of clinical endeavour, it is specially appropriate that the first professor should be a surgeon of the stature of Charles F. W. Illingworth, Regius Professor of Surgery at the University of Glasgow, a gifted teacher and well known author.

Professor Illingworth will arrive in Canada during the last week of September 1960, and will spend two weeks at the University of British Columbia. During the week of October 10, Professor Illingworth will attend the Clinical Congress of the American College of Surgeons in San Francisco, which will be followed by a two week visit to the University of Manitoba. Professor Illingworth's stay in Canada will be concluded with brief visits to The Royal College Headquarters in Ottawa and to Toronto.

In the April 1960 issue of the Canadian Journal of Surgery, the Newsletter announced that plans were being made for a Western Canada Regional Meeting of the College to be held in Saskatchewan in 1960. Subsequently it became apparent that such a meeting would conflict with other meetings planned in that area, and it therefore became necessary to transfer the 1960 Regional Meeting to Southwestern Ontario. The Southwestern Ontario Regional Meeting will be held at the University of Western Ontario and its affiliated teaching hospitals in London on November 15 and 16, 1960. Professor Angus D. McLachlin has been named Chairman of the Committee on Arrangements for this Regional Meeting, and he and his Committee are preparing an excellent programme which will be announced at a later date. An invitation is extended to all Fellows and certified specialists of The Royal College of Physicians and Surgeons of Canada living in Southwestern Ontario to attend this meeting. All qualified medical practitioners in the area will also be welcome to attend.

Of interest to many in the profession is the announcement by Council, of the election to Fellowship, under Article 3, Section 3 of the By-laws, of the following surgeons:

Dr. Fernand Montreuil of Montreal, Professor of Otolaryngology at the University of Montreal

Dr. J. V. V. Nichols of Montreal, Associate Professor of Ophthalmology at McGill University

Dr. George W. Armstrong of Ottawa, Consulting Orthopædic Surgeon at the Ottawa Civic Hospital, Past-president of the Canadian Orthopædic Association and President of the Canadian Medical Protective Association.

The Gordon Richards Memorial Lectureship for 1961 will be presented at the Annual Meeting of the College, to be held

in Ottawa, January 19 to 21, 1961. This will be the first time that this Lectureship, which is sponsored annually by the Ontario Cancer Treatment and Research Foundation, has been presented at the Annual Meeting of the College. The name of the 1961 lecturer has not yet been announced..

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary
August 19, 1960.

NOTICES

PROFESSOR F. E. BRYANS JOINS THE JOURNAL'S ADVISORY BOARD

The Editorial Board is pleased to announce that Professor Frederick E. Bryans of Vancouver, B.C. has consented to replace the late Professor A. M. Agnew on the Advisory Board, representing the specialty ot Gynæcology. Dr. Bryans graduated in medicine from the University of Toronto in 1946, and qualified by examination for fellowship in the Royal College of Surgeons of Canada (Obstetrics and Gynæcology) in 1952.

Dr. Bryans recently succeeded the late Dr. Agnew as Professor and Head of the Department of Obstetrics and Gynæcology in the University of British Columbia, Faculty of Medicine and the Vancouver General Hospital.

The Board welcomes Dr. Bryans and looks forward to his long and pleasant tenure as the Journal's adviser on matters pertaining to his specialty.



Prof. F. E. Bryans

DR. MAURICE DUFRESNE LEAVES THE JOURNAL

The Advisory Editorial Board regrets that Dr. Dufresne has resigned as assistant editor of C.M.A. publications. He has been responsible in large measure for the more recent issues of the *Canadian Journal of Surgery*. The Editorial Board decided at the outset that each article submitted for publication should be read and commented upon by two of its members in addition to the Chairman. The transmission of these comments to the authors of manuscripts without offence, is one of the more difficult duties of the editor and requires at times much diplomacy. Dr. Dufresne possessed the tact and humour needed to do this successfully. We wish him well in the new venture he has undertaken and hope he will continue to take a friendly interest in the welfare of the journal.

Robert M. Janes,
Chairman, Editorial Board

FORTHCOMING MEETINGS

UNIVERSITY OF TORONTO POSTGRADUATE COURSE IN OTO-LARYNGOLOGY

A graduate course in oto-laryngology will be presented by the Staff of the Department of Oto-laryngology on May 11, 12 and 13, 1961. They will be assisted by two distinguished guests, Dr. Philip E. Meltzer, Professor of Oto-laryngology, Harvard Medical School, and Chief of Oto-laryngology, Massachusetts Eye and Ear Infirmary, and Dr. W. G. Hemenway, Department of Oto-laryngology, University of Chicago.

The first session will begin in the afternoon of May 11, in the Royal York Hotel, Toronto, in association with the Section of Oto-laryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss. and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of this specialty in their management.

The fee for the course will be $40, and will include a complimentary dinner.

All enquiries should be addressed to the Director, Division of Postgraduate Medical Education, University of Toronto.

REFRESHER COURSE IN EYE SURGERY —UNIVERSITY OF TORONTO

The Faculty of Medicine, University of Toronto, will hold a Refresher Course in Eye Surgery from March 20 to 22, 1961. The instruction will consist of lectures, operative sessions and a special symposium on cataract surgery.

Dr. Robert N. Shaffer, University of California, San Francisco, and Mr. B. W. Rycroft, F.R.C.S., London, England, will be guest surgeons. The staff of the Department of Ophthalmology will contribute extensively.

The course will be limited to 50 members and is open to eye, ear, nose and throat specialists. Application should be made to the Director, Division of Postgraduate Medical Education, Faculty of Medicine, University of Toronto, Toronto 5, Ontario, Canada, before January 31, 1961.

On March 18 there will be a Departmental Research meeting and Dr. H. M. Burian, University of Iowa, will be guest speaker. Members of the Eye Surgery Course are invited to attend.

NEW YORK UNIVERSITY

A three day symposium on the surgery of endocrine organs will be presented by the Schools of Medicine of the New York University Medical Center from November 17 to 19, 1960. Participants will include a guest faculty of some of the most distinguished authorities in this field, among whom are Doctors Oliver Cope, Edwin Ellison, David Hume, Michael Lepore, John A. Oates, Olaf H. Pearson, Milton H. Porter, William W. Scott, Paul Wermer, Sidney Werner and others. Sessions will be under the direction of Dr. John H. Mulholland, professor and chairman, Department of Surgery, and Dr. S. Arthur Localio, professor of clinical surgery.

Surgical diseases in which the endocrine system is directly involved or in which endocrine influences are important will be discussed. Functioning tumours of endocrine structures, derangements of internal secretions that are amenable to surgical treatment, and endocrine factors in metastases of cancer are considered from physiologic and diagnostic standpoints.

A round table discussion by all the participants on Saturday morning, November 19, will conclude the symposium.

Further details and application may be obtained from the Office of the Associate Dean, New York University Post-Graduate Medical School, 550 First Avenue, New York 16, N.Y.

AMERICAN COLLEGE OF SURGEONS

The 46th Annual Clinical Congress of the American College of Surgeons will be held in San Francisco, California, from October 10 to 14, 1960.

Doctors from all parts of the United States as well as from many other countries will attend sessions at this largest meeting of surgeons. More than 1000 will take part in the various programs as authors of research reports, teachers of postgraduate courses, participants in panel discussions, lecturers, and operating surgeons in motion pictures and closed-circuit telecasts.

Major addresses will be made by Dr. I. S. Ravdin, Philadelphia, Chairman, Board of Regents and incoming President of the College; Dr. Joseph Trueta, Oxford, England, who will speak on trauma and the living cell. Dr. Wendell M. Stabley, Director of the virus laboratory at the University of California, and Nobel winner in chemistry, will deliver the Martin Memorial Lecture, named for the College founder, Franklin H. Martin, on the subject of virus-cancer relationships. Mr. Leslie Philip Le Quesne, London, England, will give the annual Baxter Lecture, speaking on body fluid disturbances resulting from stomach obstruction. On the final evening, October 14, initiates will be presented for fellowship, honorary fellowships will be conferred and officers inaugurated.

The retiring President of the College is Dr. Owen H. Wangensteen, Minneapolis. Headquarters for the meeting will be the Civic Auditorium, with some sessions scheduled at nearby hotels.

Dr. Leon Goldman, Professor and Chairman, Department of Surgery, University of California School of Medicine, Berkeley, San Francisco, is Chairman of the local committee on arrangements.

THE SOUTH WESTERN ONTARIO SURGICAL ASSOCIATION

The Annual Meeting of the South Western Ontario Surgical Association will be held on November 16, 1960 at Victoria Hospital, London, Ontario. Clinical sessions will begin at 9 a.m.

For further information write to Dr. R. M. McFarlane, Secretary, Surgery Office, Victoria Hospital, London, Ontario.

HUMBOLDT UNIVERSITY, BERLIN

The celebration of the 250th anniversary of the Charité of the Faculty of Medicine will be held in Berlin from November 6 to November

19, 1960, in connection with the 150th anniversary of the Humboldt University.

Applications for participation are to be directed to the Committee for the Preparation of the 250th anniversary of the Charité, Berlin N 4, Schumannstrasse 20-21, c/o Professor Dagobert Müller, secretary of the committee.

INTERNATIONAL COLLEGE OF SURGEONS

Southern California Chapter

The Third Western Sectional Meeting sponsored by the International College of Surgeons will be held in Las Vegas, Nevada, at the Riviera Hotel, beginning with registration on Sunday, November 20, 1960, and scientific meetings on Monday and Tuesday, November 21 and 22. A tentative programme will include presentation of 16 scientific papers.

The Riviera Hotel has offered their newest facility, a $500,000 convention hall and exhibit wing, which will accommodate 1400 persons. There is adequate space adjacent to the convention hall for scientific and commercial exhibits.

The Annual Banquet will be held on Monday evening in one of the most luxurious dining rooms of the hotel. One luncheon meeting will be devoted to a question and answer session by those presenting papers. The Women's Auxiliary will hold a luncheon-fashion show meeting for husbands and wives. Last year's convention was a great success, and it is expected that this one will be even more successful with many representatives from the 11 Western States, and also from the two new States, Alaska and Hawaii.

Further information may be obtained from the Secretary, Dr. F. M. Turnbull, Jr., M.D., 1930 Wilshire Blvd., Los Angeles 57, California.

BOOK REVIEWS

(See also pages 34, 49, 55, 64 and 81)

OSTEOCHONDRITIS DISSECANS. Loose Bodies in Joints. Etiology, pathology, treatment. I. S. Smillie, Lecturer-in-Charge, Department of Orthopædic Surgery, University of St. Andrews. 224 pp. Illust. E. & S. Livingstone, Ltd., Edinburgh and London. The Macmillan Company of Canada Limited, Toronto, 1960. $10.25.

This excellent and beautifully illustrated monograph on osteochondritis dissecans is the result of the author's interest in the knee joint and his experience with over 300 cases of this condition. The thesis is presented that lesions of similar radiological appearance can be separated into four distinct pathological entities: (1) anomalies of ossification, (2) juvenile osteochondritis dissecans, (3) adult osteochondritis dissecans, (4) tangential osteochondral fractures. All four of these are related in ætiology to the mild repetitive trauma causing osteochondritis dissecans. The relationship to abnormalities of endocrine function, local blood supply, or other constitutional factors is considered. In the juvenile variety trauma is superimposed on the ischæmic bone; in the adult group trauma produces the ischæmic bone.

The knee joint and Frieberg's infraction of the metatarso-phalangeal joint (Köhler's second disease) are the two most common sites. They are considered both from the view of ætiology and pathology in Part 1 and clinical features and treatment in Part 2. The less common sites—elbow, ankle and hip joints are of course also covered.

There is, from the introduction to the index, a logical development of the author's argument both in the text and carefully arranged numerous illustrations. This book can be highly recommended as a well reasoned account of the causes and conservative surgical management of osteochondritis dissecans.

BODY FLUIDS IN SURGERY. A. W. Wilkinson, Ch.M., F.R.C.S.E., F.R.C.S., Nuffield Professor of Pædiatric Surgery, The Institute of Child Health of the University of London. 276 pp. Illust. 2nd ed. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1960. $3.60.

The second edition of Wilkinson's valuable book on fluid and electrolyte balance was needed and is up to date. Much work has been done in preparing this edition and a better understanding of the problems of the patient with disturbed fluid balance and chemistry has led to almost complete rewriting five years after the first edition. The rather complex material and the application of these concepts to the surgical patient before, during and after his operation is presented well. The book is readable. It includes a chapter on the disturbances of fluid and electrolyte balance in infancy and childhood, for though much remains to be done in the understanding of the physiology of the newborn baby, it is not enough to regard the infant as "just a little adult".

This book is recommended unreservedly to practising surgeons and students in surgery, and especially to those interested in gastrointestinal work, severe trauma and burns.

PRACTICAL PROCTOLOGY. Louis A. Buie, Sr., M.D., F.A.C.S., Emeritus Member, Section of Proctology, Mayo Clinic, and Emeritus Professor of Proctology, Mayo Foundation. 737 pp. Illust. 2nd ed. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $24.75.

(Continued on page 142)



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(Continued from page 140)

This is the second edition of a well known text, first published in 1937. Dr. Buie is one of the founders of the specialty of proctology and his long association with the Mayo Clinic gave him opportunity to see a great variety of cases in this field. His personal experience in proctology is almost unsurpassable. This book epitomizes a lifetime career in a field to which he himself made major contributions.

The text carries all the advantages and some of the disadvantages of a book based on wide personal experience. It is not a simple repetition of other textbooks in the field. The author impresses the reader in every line with the fact that he has seen and treated even the rarest conditions and that the choice of the method proposed is based on sound clinical experience. The disadvantage of such a presentation is a slight prejudice and sometimes disregard of other opinions.

The book is a well balanced text. It starts with chapters on anæsthesia in proctology and the methods of proctoscopy and sigmoidoscopy. The embryology and anomalies of the gastrointestinal tract are well described and beautifully illustrated. The diagnosis and treatment of hæmorrhoids, fistulae, prolapse, etc. are clearly described and the operative proceedures are well illustrated. Anal pruritis is discussed in 30 pages with some very useful therapeutic suggestions. The chapter on ulcerative colitis (or, as the author likes to call it, "thromboulcerative colitis") is handled with too much stress on surgery. One may almost get the impression that medical management is always doomed to failure.

In conclusion, this is a useful book for the general practitioner and the surgeon. Physicians and gastroenterologists who read it may often disagree but it may make them understand why there is so frequently a difference of opinion between themselves and their surgical colleagues.

KLINISCHE CHIRURGIE FUR DIE PRAXIS. In vier Banden. Band 11. Lieferung 1. (Clinical Practice of Surgery. In 4 volumes. Vol. 11. Part 11). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 224 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $10.00.

The book under review is a segment of a four volume work on surgery, and deals with diseases of the chest.

The introduction contains a concise history of heart and lung surgery by Professor Dr. O. Diebold of Hamburg.

The book covers the fields of physiology and pathophysiology of respiration and circulation, as well as their function tests. These and the chapter on postoperative respiratory insufficiency are brought up to date and done exceedingly well. Further parts of the book deal with diseases of the chest wall and breast requiring surgical intervention.

In order to evaluate this book one has to understand the differences in the practice of surgery confronting the general practitioner in continental Europe and on the North American continent. In North America, the practitioner usually participates in the surgical procedure his patient is undergoing at the hospital, whereas in Europe the patient is turned over by his doctor to the hospital surgeon for operation.

Therefore, this book emphasizes the diagnostic part of diseases with all the tests involved, and selection of cases for operation, neglecting surgical techniques.

The chapter on cancer of the breast is more or less a German reprint of the same subject contained in Hamilton Bailey's excellent book "A SHORT PRACTICE OF SURGERY".

Readers of this book can look forward with interest to the publication of Volume 3 of this work dealing amongst other subjects with intestinal obstruction, and appendicitis and their surgical treatment.

For the German speaking physician taking his Medical Council of Canada examination and encountering language difficulties, this book will provide a valuable aid to preparation for his later studies of Hamilton Bailey's "A Short Practice of Surgery".

The reviewer considers that a translation of this book into the English language is not warranted.

KLINISCHE CHIRURGIE FUR DIE PRAXIS. In veir Banden. Band 111, Lieferung 1. (Clinical Practice of Surgery. In 4 volumes. Vol. 111. Part 1). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 148 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $6.70.

The authors of this book are to be lauded for their chapters on abdominal surgery. Beginning with a description of the different laboratory tests, necessarily preceding surgery of the abdomen, the authors proceed with a classification of "emergency", "urgent" and "routine" operations, stressing the need for an overall estimation of the patient's operability before surgery.

The section on fluid and electrolyte balance is covered in detail with citations of normal values, and discussion of the causes of disturbances in electrolyte balance.

The chapter on peritonitis and its operative treatment covers this subject in detail. With no intent to detract from the value of this book one need not accept without reservation the author's statement that at least 96% of all diffuse peritonitis cases have a fatal outcome unless immediate operation is performed. A more flexible approach to this problem with adjustment of the therapy in keeping with the origin of the peritonitis would seem appropriate in this regard.

The pages on hernia are well written, and readers will find a comprehensive classification of all types of herniae with diagrams and illustrations providing a ready understanding of this complex problem. Physicians dealing with herniae, resulting from accidents will be interested in the section dealing with accident and herniae, clarifying the connection between cause and result. The author establishes rules to be followed for recognition of such claims and cites Bier's six postulates.

The value of this book lies in its easy flow of uncomplicated language; a definite improvement over German textbooks of surgery of the past. Sentences are short and remarks to the point, bringing this book more in line with English publications.

It is of further interest to note that the authors quote extensively from British and North American medical literature.

A hard cover edition of the complete volume would prove beneficial to readers.

This book is highly recommended to physicians with a knowledge of the German language.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Actualités sur les Maladies des Veines. Varices, Phlébites, Hémorroides. Colloque tenu dans le service de Professeur André Sicard, Hôpital Beaujon, Paris, April 1958. 89 pp. Illust. L'Expansion Scientifique Française, Paris. 13NF.

Anti-Koagulantien in der Chirurgie. H. A. Thies and J. Oeri. 54 pp. Benno Schwabe & Company, Basel, Switzerland; Intercontinental Medical Book Corporation, New York, 1960. $1.75.

Clinical Orthopædics. Editor-in-Chief Anthony DePalma, with the assistance of the Associate Editors, The Board of Advisory Editors, The Board of Corresponding Editors. Number 16. 315 pp. Illust. J. P. Lippincott Company, Philadelphia and Montreal, 1960. $7.50.

Die Altersveranderungen der Halswirbelsaule. Ulrich Ecklin, Director of the Institute of Anatomy, Zurich University. 81 pp. Illust., Springer-Verlag, Berlin, Gottingen and Heidelberg, W. Germany, 1960. DM 28.

Die Behandlung der Schadelbasisbruche. Frontobasale und laterobasale Frakturen de Nase der Nebenhohlen und des Ohres. Prof. Dr. H.-G. Boenninghaus. Oberarzt der Université Hals-Nasen-Ohrenklinik, Frankfurt Am Main, West Germany. 200 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $9.05.

Exposés d'Anesthésiologie. A l'usage des practiciens et des étudiants. P. Huguenard et P. Jaquenoud. Première série. 218 pp. Illust. Masson & Cie, Paris. 23NF.

Exposes d'Anesthesiologie. A l'usage des practiciens et des étudiants. P. Huguenard et P.

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HISTORY OF CANADIAN SURGERY

ICELAND'S GIFT TO CANADIAN MEDICINE

ROSS MITCHELL, M.D., *Winnipeg*

Ice and Fire! These two words depict Iceland set in northern seas with its glaciers, volcanoes, lava fields, geysers and hot springs. They are reflected in the Icelandic character, patient, persevering, phlegmatic, stubbornly enduring yet eloquent, musical and capable of explosive bursts of energy. The remoteness of the land has made its people self-reliant, freedom-loving and independent; its austerity has made them work hard and venture far afield. The early Vikings founded a prosperous settlement in Greenland and were the first white people to reach North America, "Markland" and "Vinland".

Iceland has a glorious past. The first National Assembly or "Althing" met on a plain near Reykjavick in the year 930. Their golden age of poetry was in the 10th and 11th centuries when their skalds composed the Eddas and Sagas, but the tradition lingers. Illiteracy is almost unknown and the poet is revered. The population has never been large. The census of 1890 gave the total as 70,927. From 1872 to the end of the century at least 15,600, roughly one in six, emigrated to America, chiefly to Manitoba.

A group which had embraced Mormonism went to Utah in 1855, another to Brazil in 1863, a larger group under Pall Thorlakson went to Wisconsin. In 1873 a small group went to Ontario and was placed on homesteads at Rosseau in the Muskoka district. Another party went to Nova Scotia, but land selected for them was poor and almost all these settlers left the province. A majority of the Rosseau group, 385 in number, transferred to Kinmount, fifty miles north of Lindsay. Their leader was Sigtrygur Jonasson, a young man of energy and education. Life at Kinmount was hard but was made lighter by John Taylor who lived nearby as representative of the Shantymen's Union sponsored by the British and Foreign Bible Society. He told Jonasson that the federal government was offering free land in Keewatin, the unorganized district north of the new province of Manitoba. Taylor went to Ottawa and spoke to the federal ministers on migration but they were dubious of the project. Lord Dufferin the Governor General of Canada who in 1856 had visited Iceland, was sympathetic and promised support. A meeting of the Kinmount group was held on June 30 after John Taylor's return. It was decided to send a chosen few to spy out the land. Sigtrygur Jonasson, John Taylor and three others set out on July 2, 1875, and were joined by S. Christopherson of Wisconsin. They landed at Winnipeg on July 18, and after purchasing provisions were guided in a York boat by Joseph Monkman, the well known Metis of St. Peters. The six men approved the site, 36 miles in length, on the west side of Lake Winnipeg which now is between Gimli and Riverton, for these reasons: (1) There was a waterway from Winnipeg, (2) the land was reasonably good, (3) there were trees and streams and (4) the lake offered good fishing.

When the delegates returned to Kinmount a meeting was held and the great majority decided to migrate though it was late in the season. On their way to Collingwood they were joined by a party from Iceland. They crossed in an overcrowded steamer, then travelled from Duluth to Grand Forks in box cars. The editor of the *Plaindealer*, watching them embark on the sternwheeler "International", wrote

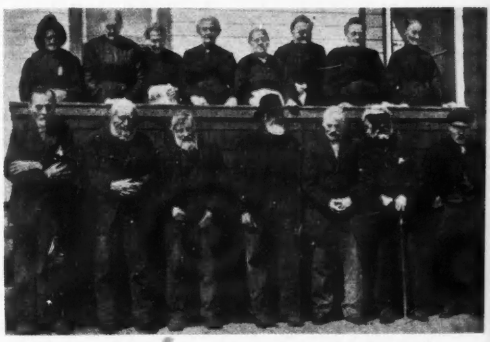


Fig. 1.—The Sunset of Life. Betel Icelandic Old Folks Home, Gimli, Man.

that they were "the finest group of emigrants he had seen" and the editor of the *Winnipeg Free Press* later re-echoed this. It was now mid-October and winter was not far off, but the band, 285 in number, decided to push on to found their own homes. In a York boat and six flatboats they drifted down the Red River, then were towed by the Hudson's Bay steamer "Colvile" to Willow Point and spent the night of October 22 there. On the next day they started to build 30 log houses, one for each stove they owned! With cheerful enthusiasm they named their first settlement Gimli, "Home of the Gods". Other settlements were made further north at Lunde (Riverton) and on Big (Hecla) Island.

New Iceland

The winter was long and exceptionally cold but in the summer the settlers were cheered with the arrival of "the big party", 1200 or more from Iceland. In September of that year, 1876, smallpox broke out, mild and undiagnosed at first, but then spreading and becoming virulent. An appeal for aid was made to Manitoba. Dr. Young and Dr. Lynch were sent out with vaccine and medical supplies. Dr. Young contracted the disease, Dr. Beddome and Dr. Baldwin came in to help, a hospital was set up at Gimli and quarantine was imposed at Netley Creek. By the end of the year the epidemic had subsided but it left over 100 dead.

The next year was not dogged with misfortune. A school, taught by a niece of John Taylor had 30 pupils at Gimli, a handwritten newspaper was circulated and verses were hung on trees as in the Forest of Arden. On September 14, 1877, the Governor-General and his party visited Gimli, inspected some farms, looked in at the homes and addressed the people. After mentioning that in some of the small houses he had seen libraries of 30 to 40 books, he said: "I trust you will continue to cherish for all time the heart stirring literature of your nation and that from generation to generation your little ones will continue to learn in your ancient Sagas that industry, energy, fortitude, perseverance and stubborn endurance have ever been the characteristics of the noble Icelandic race."

Just before his visit the first newspaper printed in Iceland appeared with the appropriate name *Framfari* or Progress. It lasted for four issues. Laws were drawn up and religious services were carried on by the Rev. Jon Bjarnson and the Rev. Paul Thorlakson. When Manitoba was enlarged in 1881, New Iceland was included in the province. Around 1882 there were migrations to Pembina County, North Dakota and in Manitoba to Winnipeg, Selkirk—where it was expected that the new railway would cross the Red River—Lundar, Arborg, Glenboro, Baldur, Swan River, Dauphin and Morden. Later settlements were established in Saskatchewan and Alberta.

The Second Generation

It was not long before the second gen-

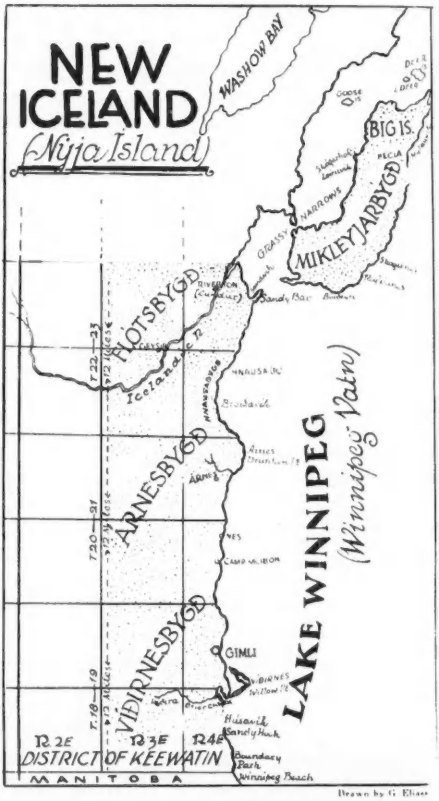


Fig. 2.—Map of New Iceland.

eration began to make its mark in the professions, literature, music, art, government and sport. In 1885 the first Icelander graduated from the University of Manitoba and by 1930 there were over 150 graduates of Icelandic descent. Skuli Johnson, late professor of Classics in the University, was the Rhodes Scholar from Manitoba in 1909 and in the next year the Rhodes Scholar was Joseph T. Thorson who went on to become Dean of the Law School, Member of Parliament for Winnipeg South, Minister of National War Services, and since 1942 president of the Court of Exchequer.

Others who have won fame in the law are Thomas H. Johnson, Attorney General of Manitoba, Mr. Justice H. A. Bergman, "one of the best legal minds in Canada", chairman of the board of governors, University of Manitoba and two, happily still living, Walter J. Lindal, county court judge, and Senator G. S. Thorvaldson.

In art one may point to Emili Walters whose pictures hang in the galleries at Glasgow, Dublin, Finland, France, Iceland and Canada; in education to the chemist Thorberg Thorvaldson, D.Sc. of the University of Saskatchewan, Thorvaldure Johnson, Ph.D., plant pathologist in the University of Manitoba, T. J. Oleson, Professor of Mediæval History in the University of Manitoba and the late Dean O. T. Anderson, United College, Winnipeg.

The outstanding living Arctic explorer is Manitoba-born Vilhjamur Stefansson, LL.D. who lives in Manitoba. Many have made names in music as performers and teachers. In sport, the Icelandic Falcons of Winnipeg won the first Olympic hockey championship in 1921 and Tom Johnson of Baldur who plays with the Montreal Canadiens won the award in 1959 for the best defenceman in the National Hockey League.

Many Icelandic girls have graced the nursing profession. The first, and one of the best known, was the late Inga Johnson who after graduating from the Winnipeg General Hospital School of Nursing, in 1907, became assistant superintendent of that school, matron of No. 4 Canadian Casualty Clearing Station, winning British and Belgian decorations and finally became superintendent of Betel Old Folks Home at Gimli.

From 1904 to 1926 there was a professorship of Icelandic in Wesley College, Winnipeg, and in 1951 a fully endowed chair of Icelandic Language and Literature was set up in the University of Manitoba.

Icelandic Doctors

The first Icelander to graduate from Manitoba Medical College, now the faculty of medicine, University of Manitoba, was Olafur Stephenson (1864-1939) who graduated in 1895 and served with the Canadian Special Hospital at Ramsgate in the Great War, 1914-1918.

Outstanding as an obstetrician was Olafur Bjornson. He came with his parents to Gimli in 1876 when he was eight years old and in going down the Red River sat on the one trunk which held the family belongings. He was educated by his father and in public schools and graduated from

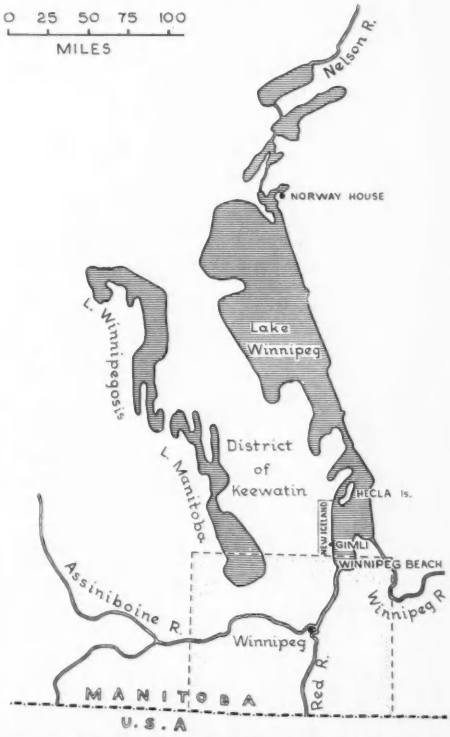


Fig. 3.—Sketch map of Manitoba and Keewatin circa 1876 showing New Iceland and Gimli.

Manitoba Medical College in 1897. After general practice he went to Chelsea Hospital, London, and the Rotunda, Dublin, in company with his close friend Brandur Brandson. On his return to Winnipeg he practised the safe and conservative methods he had learned at the Rotunda and soon was invited to teach obstetrics. He excelled as a teacher with his command of English, wisdom and humour. He published little but his method of rotating the posterior shoulder in deep arrest of the head was praised by Professor Munro Kerr in *Operative Midwifery*, 1937. He became professor of obstetrics, retired in 1932 and died October 3, 1937, much quoted and much beloved.

Brandur J. Brandson (1874-1944) also came with his parents from Iceland, but they settled in North Dakota. He was educated at Gustavus Adolphus College and graduated from Manitoba Medical College in 1900. For four years he practised at Edinburgh, N.D., then after postgraduate study with his friend Bjornson, he practised surgery in Winnipeg. From 1927 to 1934 he was Professor of Surgery and on his retirement he was made emeritus professor and the University of Manitoba conferred on him the LL.D. degree. When he travelled to Iceland in 1930 for the millenium of the Althing, he was granted an honorary M.D. degree by the University of Iceland and in 1935 the government of Iceland made him Grand Knight Commander of the Royal Icelandic Order of the Falcon. He took a great interest in his church, the First Icelandic Lutheran, and in the Old Folks Home at Gimli. Imposing in figure and gifted with oratory he was urged to enter politics but declined. A paper on hydatid disease, in which he had unrivalled experience, was written by him but not published.

Jon Stefansson (1878-1936) graduated from Manitoba Medical College and after postgraduate work in London, Vienna and Berlin became lecturer in clinical ophthalmology. He devised an operation for glaucoma. Each year an ophthalmoscope is awarded in his memory to the Manitoba medical student ranking highest in ophthalmology.

Doctors of Icelandic Descent

Baldur Olson (1888-1952) also graduated from Manitoba Medical College, served in both world wars, practised with Dr. Brandson and became widely known as a chest surgeon. He was Lecturer in Medicine from 1924 to 1932. He was appointed Medical Director of the Great-West Life Assurance Company in 1917 and retired four years later.

August Blondal (1889-1948) was born in Edinburgh, N.D., graduated from Manitoba Medical College, worked in the Royal Maternity Samaritan Hospital, Glasgow, and was Lecturer in Obstetrics, University of Manitoba 1934-1948. He was also an artist in many media, drew the design for the crest of Manitoba Medical Association and also two clever cartoons of Dr. H. M. Speechly and Dr. E. W. Montgomery.

Living Doctors of Icelandic Descent

In the living present, medical men and women of Icelandic descent are contributing to Canadian medicine. Foremost is *Paul H. T. Thorlakson*, "Thor" to his friends. The son of a distinguished Lutheran clergyman, he entered Manitoba Medical College in September 1914. He volunteered for overseas service at the end of his second year and proceeded to England as medical sergeant of the 223rd Battalion. He was returned to Canada to complete his medical education, served a year as intern in the Winnipeg General Hospital, another year in the Children's Hospital and graduated M.D.,C.M. in 1919. After postgraduate training in surgery in London and on the continent (1921-22) he entered into partnership with the late Dr. Neil John Maclean, a master surgeon, to form the Maclean-Thorlakson Clinic in 1926. In 1940, Dr. Thorlakson founded the Winnipeg Clinic. This group of 52 doctors of which he is the director, includes all the various specialties and is located in a 12 storey building.

By successive steps he rose to be surgeon-in-chief, Winnipeg General Hospital and Professor of Surgery, University of Manitoba, and since 1957, Professor Emeritus of Surgery. In 1952 the University of Mani

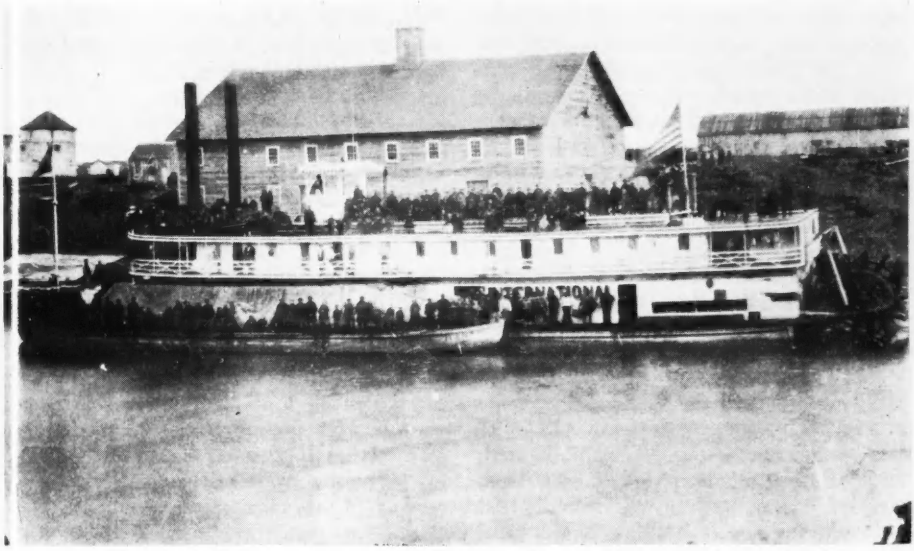


Fig. 4.—Red River steamer "International" before H.B.C. warehouse. South-east bastion of Fort Garry to the left.

toba conferred on him the degree, Doctor of Laws, *honoris causa*.

Dr. Thorlakson has been keenly interested in medical education and research. In 1938 he was appointed to the first associate committee on medical research of the National Research Council and served for eight years under the chairmanship of the late Sir Frederick Banting. At a meeting of the medical committee of the National Research Council in 1944 he recommended the establishment of a western regional conference on medical research as a postwar project. The first meeting of this group was held under his chairmanship in Vancouver in February 1945. It has met successively in the four western provinces ever since the war, giving young medical scientists an opportunity to present their work annually. This conference has been a tremendous stimulant to more active medical research in the four western Canadian universities.

In 1943 Dr. Thorlakson was a moving spirit in the formation of the Manitoba Institute for the Advancement of Medical Education and Research, with a charter membership of 36, lay and medical, and became the first president. The Institute, approved by the Board of Governors and the Medical Faculty of the University of Manitoba, has contributed over $46,000.00 to medical education and research up to the present time. In 1957 the name was changed to the Winnipeg Clinic Research Institute.

To date Dr. Thorlakson has had over 40 scientific papers published in various medical periodicals in Canada and the United States. At the present time he is president of the Manitoba Council on Education and he was chairman of the first conference held at the University of Manitoba on February 27, 1960, at which Dr. Wilder Penfield was the principal speaker.

In 1939 King Christian X of Denmark and Iceland conferred upon Dr. Thorlakson the Knight Cross of the Order of the Falcon. In 1951, seven years after Iceland had once again become an independent republic, the president and government of Iceland conferred upon him the Commander Cross of the Order of the Falcon because of his services in helping to found the first endowed chair in a university of western Canada, the chair of Icelandic Language and Literature in the University of Manitoba.

Dr. Thorlakson followed the example of Dr. Brandson and became chairman of the

Board of Directors of the Betel Old Folks Home, Gimli. Under his leadership the Home has recently been renovated and enlarged to accommodate 100 residents and also provides a 24 bed infirmary.

An example of his remarkable foresight is seen in his organization of the Manitoba Medical Centre in 1943, approved by the Provincial Legislature in 1945. At present the Medical Centre contains the Winnipeg General Hospital, the Children's Hospital, the Central Tuberculosis Hospital, the Women's Pavilion, Medical and Dental Faculty Buildings, the Manitoba Cancer Foundation, nurses' residences and residences for interns, the L. G. Bell House for married interns and the Medical Centre apartments for General Hospital staff. The new Rehabilitation Hospital has been started, a cancer hospital will be built, and a seven storey service building of the Winnipeg General Hospital is under construction. Dr. Thorlakson is the present chairman of the Manitoba Medical Centre Council.

Dr. Thorlakson's interests are not limited to Canadian medicine. He is a Member of the Royal College of Surgeons of England, a Senior Fellow of the American Surgical Association, a senior member of the Western Surgical Society, a Fellow of the American College of Surgeons, of which he was governor, 1955-58, and a member of the executive committee of the Committee on Cancer. He has held many positions in the National Cancer Institute of Canada and was president of that organization in 1951-52. He is a Fellow of the Royal College of Surgeons of Canada, member of the Canadian Association of Clinical Surgeons (western division), and President of the Medical Alumni Association of the Winnipeg General Hospital. In 1955-56 he was Honorary President of the University of Manitoba Alumni Association. In his spare time he interests himself through serving as chairman or member of committees dealing with postgraduate studies, Manitoba Medical Service, Manitoba Heart Foundation, the Committee on Rehabilitation and the 1950 National Flood Relief Committee.

Following in Dr. Thorlakson's footsteps are his twin sons, Dr. Kenneth and Dr. Robert both M.D., Manitoba 1950, wh are also members of the Winnipeg Clini and hold the Fellowship of the Royal Co lege of Surgeons of England.

George Johnson, M.D., Manitoba, 195 was called in June 1958 from practice a Gimli to be Minister of Health and Publ Welfare in the Roblin government. Sinc then he has introduced legislation whic very recently won praise for his imagin tion and enthusiasm from the forme premier, Douglas Campbell, now leade of the opposition.

To the Minister's credit are:

(1) Provision for free injection of Sal vaccine to all under 40 years of age an of a booster dose for school children. In 1959 the only deaths from poliomyelitis in Manitoba were those of two men, neither of whom had received Salk vaccine;

(2) the Social Allowances Act which provides for government aid where need can be shown;

(3) the Elderly Persons Housing Act whereby a housing project at the community level will receive government aid of one-third of the cost of construction.

(4) the Home Care Polio Programme;

(5) the establishment of a Directorate of Alternative Care with categorization of nursing homes to relieve the shortage of hospital beds, and covering the broad field of care of the elderly, the sick elderly, and the infirm elderly and chronically ill, from the acute hospital care stage through to home care;

(6) the Northern Health Services Branch with headquarters at The Pas, with Dr. P. G. Lommerse D.P.H. as Director, having under him four nurses and sanitary officers.

(7) In April 1960, a community development project was set up at Norway House and Camperville, Duck Bay, t assist Indians and Metis to find employment.

(8) Manitoba's Social Allowances Ac provides for the basic necessities of healt and the well-being of those in need. Any one within the scope of the Act may ob tain a Medicare certificate. The holder o such a certificate and the dependent mem bers of the family are entitled to the serv

ices of a doctor of their choice for office and home calls. Care in hospital is provided through the Manitoba Hospital Services Plan originated by the Manitoba government.

Larus Sigurdson, M.D., Manitoba 1927, M.A., Stanford 1928, is Associate Professor of Anatomy in the University of Manitoba. For 20 years he has been Corps Surgeon of the St. John Ambulance Society. When the Chair of Icelandic Language and Literature was set up in the University of Manitoba he was a member of the foundation committee.

Edward Johnson, M.D., Manitoba 1927, is Provincial Psychiatrist and until recently was superintendent of Selkirk Mental Hospital. In 1953 he received an award from the American Psychiatric Association for special accomplishment in administration. From 1958 to 1959 he was President of the Manitoba Medical Association.

Eyjolfur Johnson, M.D., Manitoba 1926, F.R.C.S.(Edin.) practises general surgery in Selkirk and was President of the Manitoba Medical Association, 1950-51.

H. T. Thorlakson, M.D., Manitoba 1926, is practising ophthalmology in Seattle, Washington.

K. I. Johnson, M.D., Manitoba 1937, practises in Pine Falls and is known as a cattle breeder. Recently he won the General Practitioner postgraduate award offered by the Winnipeg Clinic Research Institute.

Harold Blondal, B.Sc., E.E. 1939, M.D., 1949, studied for a year with the Atomic Energy Commission at Chalk River and a year at the Royal Cancer Hospital, London, then returned to serve as Lecturer and Assistant Professor of Physiology and Research at his *alma mater*. Recently he moved to Montreal to do cancer research with a leading pharmaceutical firm. He is son of the late A. Blondal, M.D.

John Athalstein Bildfell, M.D., Manitoba, 1933, is known for his work with the Eskimos. He is now in industrial medicine in Montreal.

One of the veteran members of the Manitoba Legislature is *Stein Olafur Thompson*, M.D., Manitoba 1921, of Riverton. He represented Gimli constituency from 1945 to 1958, winning great respect.

Brandur Thomas Hermann Marteinsson, M.D., B.Sc.(Med.) Manitoba 1921, practises general surgery in Vancouver, B.C.

Eggert Thomas Felsted, M.D., Manitoba 1944, is now practising as a radiologist in Santa Barbara, California.

Two doctors are renowned for their poetry. *Sigurd Julius Johanneson* 1867-1958, not only carried on medical practice but was editor of the weekly newspaper *Logberg* and wrote three volumes of poems, many for children. In his presidential address to the Winnipeg Medical Society, Dr. David Swartz included Dr. Johanneson among the "medical truants", a distinguished group. *Svein Ericksson Bjornson* of Arborg and Winnipeg has written fine poetry.

This list of doctors of Icelandic descent is far from complete; it makes no mention of many others who have given active service and through *"that best portion of a good man's life, his little nameless unremembered acts of kindness and of love"*, have gained the respect and affection of their communities. Canadian medicine has been enriched with the contributions of the sturdy Icelandic race and their descendants to the third generation.

ACKNOWLEDGMENTS

To Dr. Thomas A. Lebbetter, Dr. L. A. Sigurdson, Dr. D. Swartz, Dr. E. Johnson, Mr. J. T. Beck and Mr. E. S. McInnes, I am indebted for helpful suggestions. Source material has been found in: Professor T. J. Oleson's revision of Mr. W. J. Kristjansson's book *"The Icelanders in Manitoba"* still in typescript; the communications to the Manitoba Historical Society from Mr. Sigtrygur Jonasson the first Icelandic settler in Canada and Mrs. (Dr.) A. N. Sommerville; *"Medicine in Manitoba"*; the *Encyclopædia Britannica;* the *Encyclopædia Canadiana; The Manitoba Medical Review; The Canadian Medical Association Journal* and the *Manitoba Medical Register*. The extract from the Governor General's speech at Gimli in 1877 is taken from *"My Canadian Journal"* by the Marchioness of Dufferin and Ava.

The sketch of Dr. P. H. T. Thorlakson is based on the citation given by Dean Lennox Bell when he presented Dr. Thorlakson for honorary membership in the Winnipeg Medical Society.

The map for reproduction in Fig. 3 was supplied by courtesy of the Department of Surgery, Faculty of Medicine, University of Manitoba. Fig. 1 is reproduced from Dr. D. A. Stewart's book *"Home of the Happy Sunset"* with the kind permission of the author's son, Dr. D. B. Stewart. Fig. 4 is taken from a photograph in the Archives of Manitoba.

ORIGINAL ARTICLES

VENTRICULAR SEPTAL DEFECT: AN ANALYSIS OF 70 CASES IN CHILDHOOD SURGICALLY TREATED*

W. T. MUSTARD, M.D. and G. A. TRUSLER, M.D., *Toronto*

DURING THE past five years the use of the artificial heart-lung machine to bypass the heart has met with astonishing success. At the present time, most surgeons familiar with the use of a pump-oxygenator are confident of the ability of the apparatus to sustain the patient's circulation for periods up to an hour and one-half with very little deleterious effect in the postoperative period due to the use of the pump-oxygenator. This valuable tool enables both the cardiologist and the surgeon to explore the possibility of correcting defects which had hitherto been unassailable.

A classic example is the isolated ventricular septal defect and an analysis of our cases forms the basis of this report. With our medical associates, Dr. John Keith and Dr. Richard Rowe, we have been conservative in the approach to correction during these formative years.

SELECTION OF PATIENTS

The natural history of an infant or child with a ventricular septal defect has undergone close scrutiny. It appears that ventricular septal defect is the commonest congenital malformation and occurs in 22% of patients with congenital heart disease at the Hospital for Sick Children, Toronto. It also appears that small defects are compatible with longevity, and indeed small defects may close spontaneously during childhood. Larger defects are usually accompanied by an enlarging heart and over the course of years the development of pulmonary hypertension.

While mortality tables show the highest incidence to be in the first year of life, a number of infants can be carried over the first year, and indeed seem to improve on medical therapy. In our selection of p - tients for operation, we have been guid l by the clinical course of the patient, roen - genograms of the heart and lungs, the ele - trocardiograph and cardiac catheterizatio . We consider as suitable candidates f r operation children who have a large sept l defect, with a large flow to the lungs s demonstrated by a greater than 10% ste - up in oxygen saturation on passing fro a the right auricle to the right ventricle, an l with increased lung vascularity. Further - more, we feel that the optimum age for surgical correction should be under six years. Those patients demonstrating a large flow, and some degree of pulmonary hypertension, in excess of 70% of the systemic blood pressure, we consider operable if the pulmonary hypertension appears to be due to flow and not to increased pulmonary vascular resistance.

Inoperable patients are those whose pulmonary vascular resistance is such that a right to left shunt through the ventricular septal defect is present. Thus the majority of our operative cases are children between the ages of two and six years who have some enlargement of the heart, increased vascular markings by radiography, left ventricular or combined hypertrophy in the electrocardiograph, and a rise in oxygen saturation on passing from the right auricle to the right ventricle of 10% to 15% or over (Figs. 5 and 6).

OPERATIVE TECHNIQUE

We have used the Cowan pump (Fig. 1) for a good many cases, and during the pas year we have used the DeBakey type o pump, changing over simply because th Cowan pump was worn out after 10 yea of faithful service. The disadvantage o the Cowan pump was in the preparation o the bulbs, which was tedious. We have con sistently used bubble oxygenation, in ou early cases the DeWall helix type, an

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Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, January 1960.

more recently the Cooley type of oxygenator. Our approach has been through the split sternum in the majority of cases, although we have used bilateral thoracotomy, particularly in the presence of a patent ductus arteriosus. The arterial and venous pressures are monitored from the left brachial artery and vein, and the arterial inflow has been through the femoral artery. Large Bardic catheters are inserted through two separate incisions in the right atrium and outflow rates have been consistently over 70 c.c./kilo./minute. We allow the temperature of the patient to fall during the flow to approximately 30° to 32° C. We have not as yet employed a heat exchanger.

Classification of Defects

In our series of 70 cases, we have consistently attempted to measure the size of the defect and to locate it anatomically. We have found unsatisfactory most pathological classifications of ventricular septal defects and have decided to classify them embryologically. Those defects which are superior to the crista supraventricularis we call bulbar defects, in view of the embryology, and of these we had only two. Defects within the membranous portion of the septum are common and comprised 62 of the septal defects encountered. Defects of this portion are either high (anterior) or low (posterior) or a combination of the two extending beneath the septal leaflet of the tricuspid valve. Defects of the muscular septum were encountered in six instances, three of which were huge, almost single ventricle type of defects (Fig. 2). In regard to size we have classified them as large and moderate, the latter being from 1 cm. to 2 cm. In our series we had 46 large and 24 moderate defects. Of the 70 patients in this analysis, 32 had right ventricular pressures exceeding 70% of the systemic pressure.

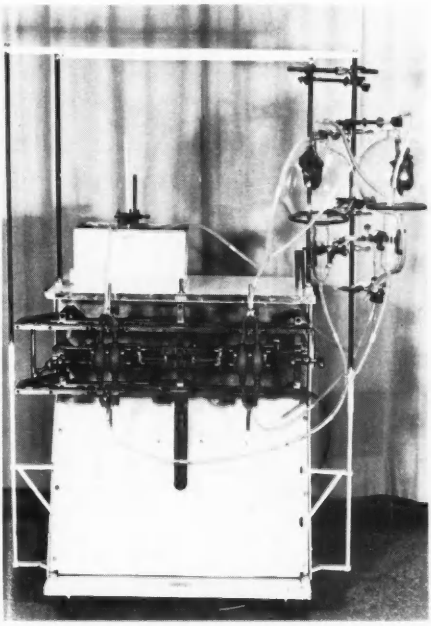


Fig. 1.—The Cowan pump and monkey lung oxygenator first used clinically in 1951.

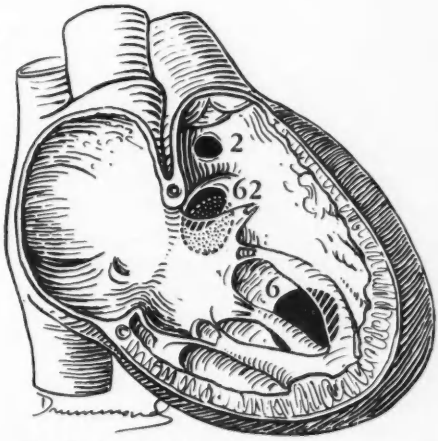


Fig. 2.—The location of septal defects as seen from the right ventricle in 70 cases.

We have excluded cases in which repair of the septal defect was accompanied by reconstruction of the pulmonary outflow tract (tetralogy of Fallot).

Technique of Closure

The method of closure has been of some concern to us, and it is rather infrequent to find a nice fibrous rim which will permit direct suture. We have consistently attempted to close by direct suture where possible, using many fine sutures of 00 or

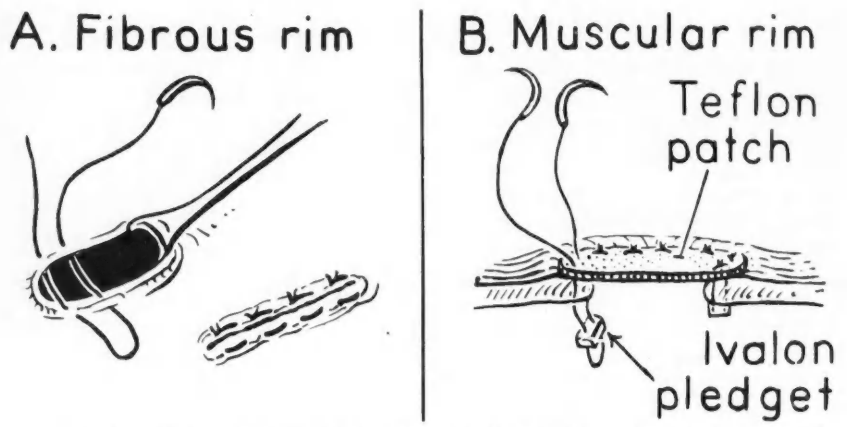


Fig. 3.—Methods of closure of the defect. On the left direct suture, and on the right a teflon prosthesis.

0000 silk, occasionally buttressed by an Ivalon button (Fig. 3). We have used Ivalon or Teflon patches in about 50% of the cases. When an Ivalon or Teflon patch is used a double ended suture is placed in the rim of the defect as illustrated in Fig. 3. We have attempted to use a triangular shaped patch of Ivalon on two occasions in the hope that the left ventricular thrust would snug this up against the defect and allow one to omit placing a suture at the dangerous area of the bundle as seen in Fig. 4.

Table I lists the associated defects encountered in this type of surgery and Table II the complications which we felt contributed to the death of 15 children. A combination of two or more complications we believe is a death warrant.

Cardiac failure, we consider is related to cardiac arrest produced either by the Melrose technique, acetyl choline or anoxia. As a result, in the last 12 cases in this series we have left the heart beating and carefully monitored (by ECG) while placing sutures in the rim of the defect. With increasing experience the need for cardiac arrest decreases.

Complete heart block is a catastrophe which must be avoided; otherwise the child will die in the postoperative period

TABLE I.—Associated Cardiovascular Conditions

Patent Ductus Arteriosus	4
Atrial Septal Defect	3
Multiple Ventricular Septal Defects	3
Pulmonary Stenosis—Valvular	2
—Infundibular	1
Cardiac Failure	2
Aortic Regurgitation	2
Mitral Regurgitation	1
Tricuspid Regurgitation	1
Coarctation of Aorta	1
Coarctation of Left Pulmonary Artery	1

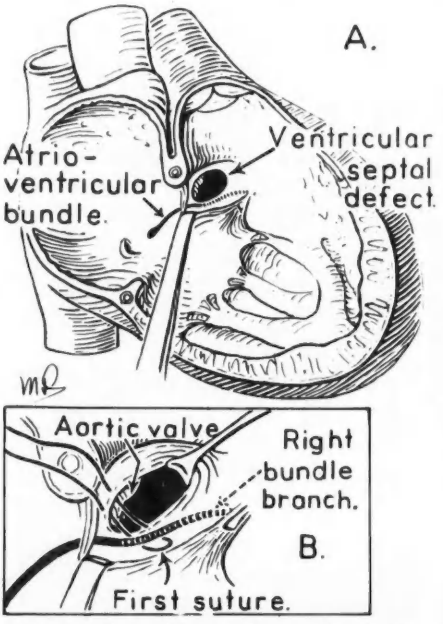


Fig. 4.—The position of the bundle of His s seen from the right ventricle.

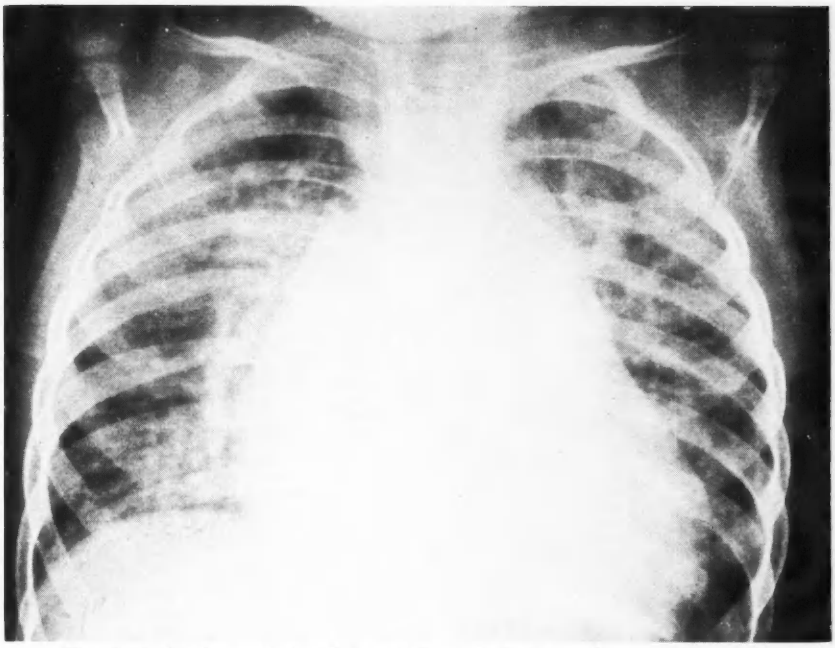


Fig. 5.—Isolated ventricular defect with marked increased vascularity of the lungs owing to increased flow.

(despite a pacemaker) as four of our patients did, or be converted into a cardiac invalid, as are our three surviving children with complete block. Their ultimate fate will probably be sudden death in a Stokes-Adams attack. In order to avoid this complication we are taking a great deal more care in placing sutures (Fig. 4), and, as noted above, closing the defect with the heart in sinus rhythm.

Persistence of the defect can be obviated by improved technique, and of course by experience. We have 10 patients with persistent defect who are greatly improved and who probably will not have to be reoperated upon. We have reoperated upon two patients successfully and one other patient unsuccessfully. However, incomplete repair, despite improvement, leaves no room for complacency and we must constantly improve our operative technique.

TABLE II.—Complications Contributing to Death (15 Patients)

Cardiac Failure		5
Complete Heart Block		4
Persistent Shunt		4
—persistent defect	2	
—missed defect	2	
Hæmorrhage		2
Atelectasis		1
Cerebral Embolism		1
Technical Error		1
Pulmonary Hypertension (late death)		1
Pump deaths		7
—pulmonary	4	
—cardiac	3	

Hæmorrhage is now not a great factor. Careful hæmostasis, adequate drainage, including chest drainage when a split sternum incision is used, and the use of antihæmophilic globulin as well as Polybrene have been responsible for this improvement.

Valvular incompetence, either tricuspid or aortic, can be avoided by meticulous care during repair. We have three patients who have some degree of tricuspid or aortic insufficiency, and these were in early cases when confidence in the pump oxygenator was not as great as it is now. One patient with aortic insufficiency has been successfully reoperated upon. On exposure of the aortic valve from above, a perfora-

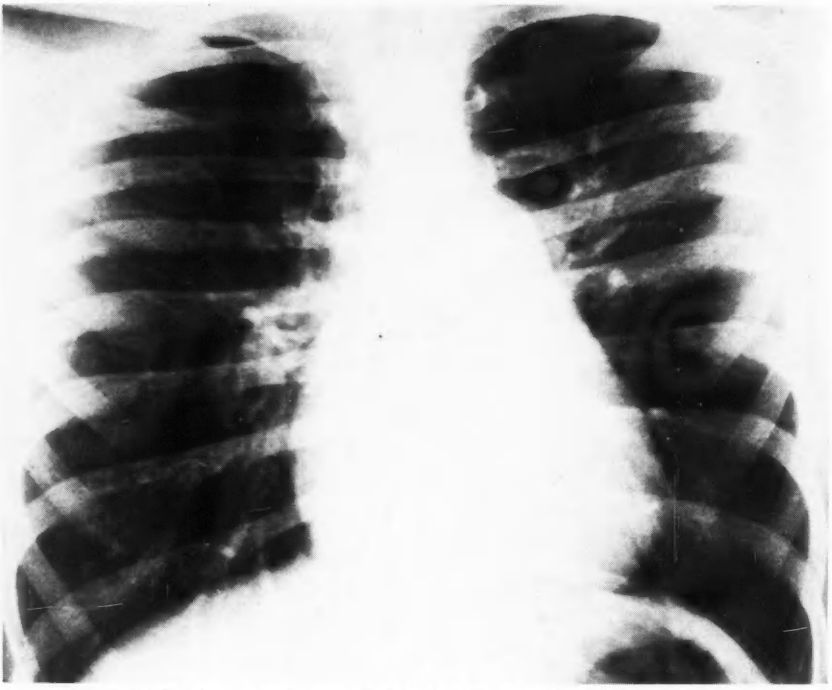


Fig. 6.—Isolated ventricular septal defect with clear lung fields owing to increased pulmonary resistance.

tion in the right coronary cusp was closed.

The use of the ventilator early, particularly in the hypertensive cases, aids in preventing atelectasis. Cerebral embolism probably occurred on the basis of antifoam improperly used in early cases.

Summary

Seventy cases of isolated ventricular septal defect have been analysed. The causes of death in 15 patients have led us to adopt the following policies:

1. No cardiac arrest.
2. Care in placing sutures near the bundle of His.
3. Longer operating time on bypass.
4. Complete closure of defect.
5. Careful hæmostasis and the use of antihæmophilic globulin.
6. Meticulous postoperative care.

Although our results must be improved it has been gratifying that in the last 35 cases of this group there have been only three deaths.

Résumé

La communication intraventriculaire constitue 22% des anomalies congénitales du cœur au "Hospital for Sick Children" de Toronto. Les communications de faible calibre sont campatibles avec une longue vie et peuvent même s'oblitérer spontanément durant l'enfance. Celles de plus grande dimension s'accompagnent habituellement d'hypertrophie cardiaque qui se complique, avec les années, d'hypertension pulmonaire.

Sont considérés pour l'intervention, les enfants de moins de six ans qui présentent une large communication avec début important aux poumons et vascularisation pulmonaire augmentée. Sont jugés inopérables, les enfants dont la résistance pulmonaire vasculaire est telle que la circulation intraventriculaire se fait de droite à gauche.

Soixante-dix patients ont été opérés. Les causes de décès chez 15 de ces malades nous ont incité à tenir la politique suivante:

1. Pas d'arrêt cardiaque.
2. Application en plaçant les sutures près du faisceau de His.
3. Temps opératoire prolongé par circulation extra-corporelle.
4. Fermeture complète de l'orifice.
5. Hémostase soignée et utilisation de globuline anti-hémophilique.
6. Soins post-opératoires méticuleux.

Les 35 derniers cas opérés n'ont produit que trois mortalités.

CHOICE OF OPERATIONS FOR URETEROPELVIC OBSTRUCTION: REVIEW OF 385 CASES*

ORMOND S. CULP, M.D.,† *Rochester, Minnesota*

WHEN RENAL function is excellent, calyces are well preserved and the patient has no symptoms, pyuria or calculi, it is foolhardy to tamper with ureteropelvic obstruction. Many patients have been observed at yearly intervals for more than a decade with no demonstrable changes in their mildly obstructed kidneys.

On the other hand, sundry combinations of pain, infection, calculi and progressive pyelocaliectasis make surgical intervention mandatory. One must then choose between nephrectomy, nephrostomy and various types of pyeloureteroplasty. This can be extremely difficult. Many obvious factors and other less tangible ones must be weighed differently in comparable clinical situations. It is unlikely that the choice of operations can ever be based on arbitrary rules, rigid prerequisites or didactic formulas. Each case promises to have its special peculiarities.

From September 1950, to March 1960, Drs. DeWeerd, Utz and the author performed 411 operations on 385 patients because of ureteropelvic obstruction. These included 185 nephrectomies, three preliminary nephrostomies, and 223 pyeloureteroplasties.

GENERAL ANALYSIS

When these patients were first seen, the gravity of their renal status varied tremendously. Some were azotemic. Others had uncontrollable infections. Many had mild symptoms but severe hydronephrosis.

In 44 cases (11.4%), only one kidney was capable of sustaining life for the reasons listed in Table I. Four of the 15 hydronephrotic sacs had to be removed under emergency conditions before the contralateral ureteropelvic obstruction could be corrected. Traumatic rupture was responsible for one of these. The other three patients had perinephric and psoas abscesses.

TABLE I.—ONLY ONE USEFUL KIDNEY

Status of other kidney	*Patients*
Agenesia	10
Hypoplasia	8
Removed elsewhere because of ureteropelvic obstruction	11
Severe hydronephrosis	15
Total	44 (11.4%)

There was bilateral ureteropelvic obstruction in 78 cases (20.3%). Treatment to date is summarized in Table II. Some of the patients who have had unilateral pyeloureteroplasty will require similar operations on the other side, while others should have contralateral nephrectomy in the near future. The 19 patients who have had nephrectomy only may never require treatment of their mildly obstructed remaining kidney, but these are being observed carefully.

Undoubtedly some of the kidneys that were subjected to plastic procedures would have been sacrificed if there had been a normal one on the other side. Extenuating circumstances left very little therapeutic latitude in 30% of the cases.

Patients represented virtually all ages from two months to 77 years. The age distribution of the 209 men and 176 women

TABLE II.—BILATERAL URETEROPELVIC OBSTRUCTION

Treatment		*Patients*
Bilateral pyeloureteroplasty		10
Both sides operated on at the Mayo Clinic	8	
One side operated on elsewhere	2	
Unilateral pyeloureteroplasty		49
Opposite side: nephrectomy	19*	
Opposite side: nephrostomy	1	
Opposite side untreated	29	
Nephrectomy—other ureteropelvic obstruction untreated		19
Total		78 (20.3%)

*Eleven performed elsewhere.

*Read at the meeting of the Canadian Urological Association, Banff, Alberta, July 1960.

†Section of Urology, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. The Mayo Foundation is part of the Graduate School of the University of Minnesota.

TABLE III.—Age and Sex

Years	*Men*	*Women*	*Both sexes*	*Percentage*
1/6-9.....	20	9	29	7.5
10-19.....	22	17	39	10.1
20-29.....	29	22	51	13.3
30-39.....	37	41	78	20.3
40-49.....	42	36	78	20.3
50-59.....	37	30	67	17.4
60-69.....	18	19	37	9.6
70-77.....	4	2	6	1.5
Total.....	209	176	385	100.0

is shown in Table III. It is noteworthy that about 40% of the patients were first seen when they were 30 to 50 years of age.

There was no significant difference in the incidence of right and left ureteropelvic obstruction.

Nephrectomy

When one kidney is functionless and the other appears normal, nephrectomy is the obvious procedure of choice. One kidney in this series contained 8000 c.c. and two held more than 5000 c.c. When an unexpected neoplasm is encountered, as was true in three instances, and the opposite kidney is capable of sustaining life, the wisdom of nephrectomy is apparent. If the kidney is studded with cortical abscesses, as were seven in this series, nephrectomy seems prudent. If leukoplakia promises to compromise the result, as in three of our patients, the kidney probably should be removed. Many unique situations, including bizarre anomalies, may indicate clearly that nephrectomy is the procedure of choice despite reasonably good renal function.

TABLE IV.—289 Patients with Normal Contralateral Kidney

Age in years	*Treatment*			
	Pyeloureteroplasty		*Nephrectomy*	
	Patients	*Percentage*	*Patients*	*Percentage*
1/6-9	11		7	
10-29	40	80.4	25	58.9
30-49	60		57	
50-59	22		32	
60-69	5	19.6	26	41.1
70-77			4	
Total	138 (47.8%)*	100.0	151 (52.2%)*	100.0

*Percentage of 289.

When the benign hydronephrotic kid ey seems to have equivocal value, decisi ns can be truly soul-searching. Unfortunat ly, there are no infallible indices of the al- vageability of kidneys. Yet saving the pa- tient is infinitely more important than s v- ing nephrons. Two seldom mentio ed factors warrant serious consideration w en the patient has a good contralateral kid ey.

1. *Rehabilitation.* — Too frequently ir- geons seem to forget that the pat nt should again be able to become a us ful citizen. Executives and office workers do not have to face the same physical le- mands as most labourers. Repeated ho pi- talization can mean economic disaster to many families. The poor man's urinary tract should not be "sold short" because of his social or financial position, but surgeons must try to look further ahead than the day John Doe can be dismissed from the hospital. Technical triumphs do not necessarily assure the most practical results. Multiple operations on the same kidney have created an appalling number of "urologic cripples". Primary nephrectomy will continue to be the most conservative procedure in many instances.

2. *Age.* — Although chronologic age can be woefully deceiving, one should be extremely reluctant to perform a plastic operation if the patient is more than 60 years of age. Indeed, doubts can be raised about the justification of such procedures after the age of 50, if the other kidney has good function. Infections may be deep seated and refractive, and the risk of secondary nephrectomy in the older age groups should not be ignored.

The influence of age in the choice of operations in this series is pointed up by the 289 patients who had a normal kidney on the opposite side, despite the fact that not all of the diseased kidneys had the same functional potential (Table IV).

Although there were many more pyeloureteroplasties than nephrectomies in the entire series, more kidneys were removed than were saved in the unilateral group. In patients of 50 years or older there were more than twice as many nephrectomies as pyeloureteroplasties.

Nephrostomy

Timely nephrostomy can be a lifesaving measure, but the practicality of preliminary nephrostomy alone in cases of ureteropelvic obstruction is extremely limited.

Even though prompt and free drainage may be of paramount importance in many instances, secondary operations on kidneys are notoriously difficult. Excessive inflammatory reactions after the initial procedure can make the subsequent technique of preference a mechanical impossibility.

Unless the patient's general condition is most precarious, it is wise to correct the obstruction during the primary operation. Usually this requires relatively little more operating time, and it is unlikely that the surgeon will ever have a better opportunity to foster a good result.

There is one conspicuous exception to this dictum. Occasionally, a kidney that is not expendable is so huge that anatomic relationships are badly distorted. No type of plastic operation will be feasible until the pelvis shrinks and appropriate deployment of tissues can be made properly and precisely. Even extensive resection of the renal pelvis will not always suffice.

Five patients had nephrostomies before they were referred to the Mayo Clinic. The justification of these procedures is not questioned, for subsequent pyeloureteroplasties were deferred until six months to four years later.

Preliminary nephrostomy drainage was employed for only three of the untreated hydronephrotic kidneys. Each of these had to be preserved and each contained more than 1000 c.c. Two of the obstructions were eliminated during the subsequent two years. The third has been observed for eight months and the concentration of the patient's blood urea has dropped from 339 mg. % to 48 mg. %.

Pyeloureteroplasties

Numerous types of pyeloureteroplasty have been proposed, but most of these have fallen into disrepute for many reasons. Today it is generally agreed that a truly acceptable operation should create a new transition between the pelvis and the ureter with three cardinal features: (1) Adequate calibre, (2) dependent position and (3) funnel shape. Failure to fulfil all three of these criteria is likely to permit some degree of continued stasis in the renal pelvis that will compromise the final result.

It is generally conceded that no one type of pyeloureteroplasty will suffice in all cases. Anyone who elects to treat ureteropelvic obstructions must be prepared to cope with four basic situations that pose dissimilar technical problems: (1) High insertion of the ureter on the pelvis, (2) junctures that already have a dependent position, (3) multiple or unusually long ureteral constrictions and (4) postoperative stenosis. Choice of operations for each of these categories offers as much individuality as the selection of a club for a particular shot on a favourite golf course. The wood or iron best suited to one man's ability may be woefully inadequate for his colleague. Nevertheless, there are limits to this flexibility if one ever hopes to shoot par.

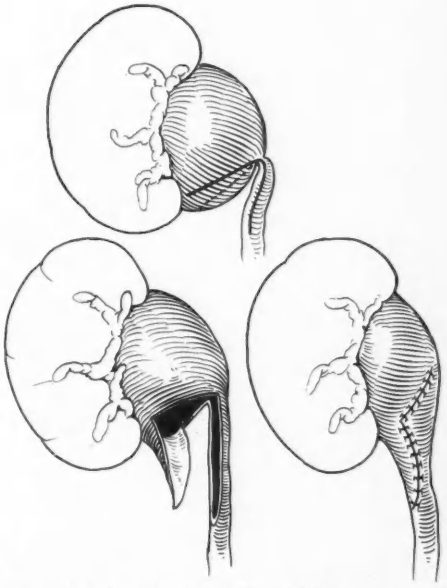


Fig. 1.—Type 1 obstruction. High insertion of the ureter on the renal pelvis; correction by Y-plasty. Incisions are made on the anterior and posterior surfaces of the dilated pelvis and along the lateral aspect of the ureter, thereby creating a flap of pelvic tissue which is sutured to the incised ureter in the form of a V.

High Insertion of Ureter

When the ureter has a high insertion on an enlarged extrarenal pelvis, as shown in Fig. 1, the well-known Foley Y-plasty has been preferred. The conversion of a Y-shaped incision to a V type of closure was not new, but Foley was the first to project this concept into a three-dimensional operation. When performed properly the Y-plasty creates a V flap that literally falls into its new position.

Unfortunately, the conventional Y-plasty has been abused. Many individuals have failed to appreciate its fundamental mechanical principles. One limb of the Y must be on the *anterior* surface of the pelvis and the other on the *posterior* surface. Both limbs must be generous to create a truly dependent V-shaped flap. The tail or stem of the Y must be on the *lateral* aspect of the ureter and carried to a point well below the lower limit of the extrarenal pelvis.

Dependent Ureteropelvic Juncture

Many obstructed ureteropelvic junctures already occupy a dependent position as shown in Fig. 2. The Y-plasty is not feasible for obvious reasons. Dissatisfaction with other plastic procedures in cases of this type prompted the evolution of a simple technique that incorporates some of the commendable features of the three-dimensional Foley operation. The cardinal steps in this method are illustrated in Fig. 2. The converging incisions are slightly longer than the ureteral constriction and they follow the spherical contour of the pelvis to avoid undesirable angulation at the base.

Multiple and Unusually Long Constrictions

Some constrictions may be far too long to be amenable to any type of revision of existing tissues (Fig. 3(a)). Problems of this type have been managed with Davis' intubated ureterotomy. However, intubation alone sometimes still leaves an abrupt transition from the pelvis to the ureter. To ensure adequate funnelling, intubated ureterotomy is combined with the spiral-flap procedure (Fig. 3(b)) or with the Y-plasty (Fig. 3(c)).

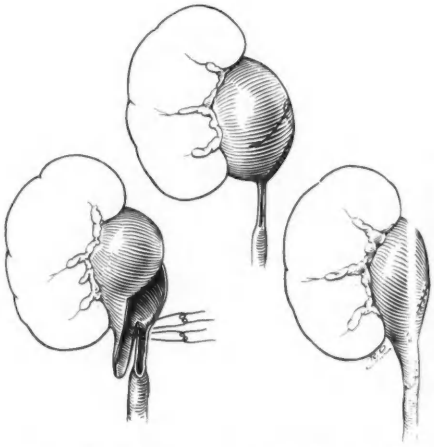


Fig. 2.—Type 2 obstruction. Ureteropelvic juncture already has a dependent position; correction by spiral-flap operation. Converging incisions from a broad, slightly oblique base adjacent to the original juncture are joined after following the spherical contour of the dilated pelvis for sufficient distance to assure a flap longer than the constricted segment of ureter. This flap is interposed between the edges of the split ureter, and the defect in the pelvis is closed.

Postoperative Stenosis

The most difficult problems were in patients who already had been subjected to some type of renal operation and in whom the ureteropelvic region was encased in scar tissue (Fig. 4). If the pelvis and ureter can be freed adequately, one of the previously mentioned techniques is preferred. But too often it is necessary to excise the entire fibrotic part. Reimplantation is the only practical solution. The manner in which it is accomplished will depend upon the amount of ureter that can be freed for the anastomosis.

Every effort should be made to create a funnel between the pelvis and the spatulated upper end of the ureter (Fig. 4(a)). Unfortunately there are occasions when one can do little more than approximate the end of the ureter and the dependent portion of the fixed pelvis (Fig. 4(b)). These are most disheartening situations, and results frequently leave much to be desired.

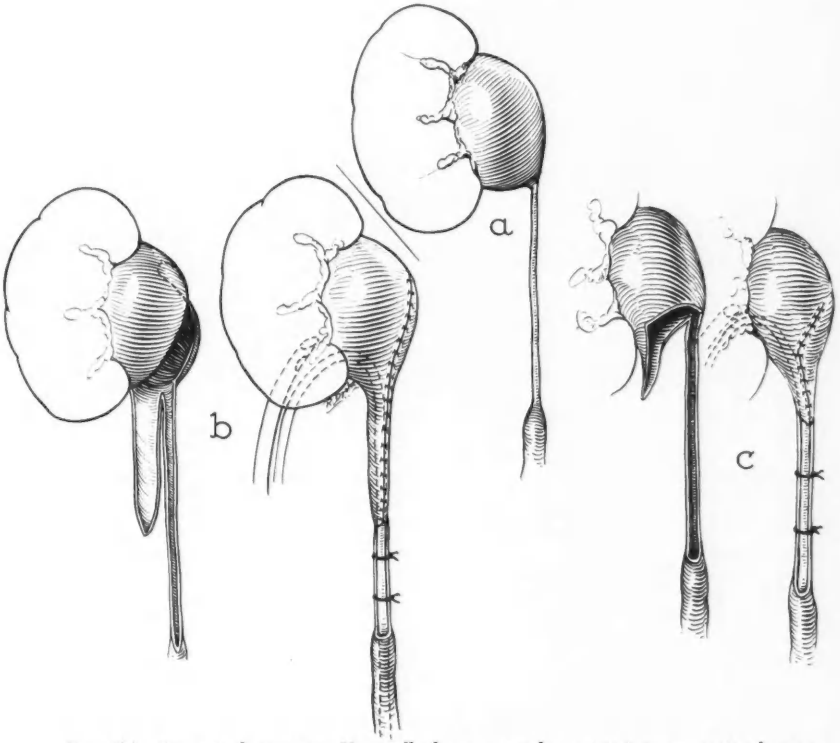


Fig. 3(a).—Type 3 obstruction. Unusually long ureteral constriction; correction by combining intubated ureterotomy with (b) spiral-flap operation or (c) Y-plasty.

Of the 223 pyeloureteroplasties in this series, 97% conformed to the four above-mentioned types.

Other Situations

In seven cases, unique situations were encountered and unorthodox methods were employed.

Obstruction confined to the lower segment of a duplicated pelvis was corrected by joining the two pelves and partially duplicated ureters (Fig. 5).

A solitary, grotesque, "cake" kidney had an obstructed ureteropelvic juncture high in the renal sinus. Some of the cortex had to be resected to permit anastomosis of the lower portion of the pelvis and the ureter. The operation failed and nephrostomy drainage was continued. A similar procedure was used with an excellent result on one half of a horseshoe kidney.

A simple Heinecke-Mikulicz procedure was tried in two early cases. It failed in one and was successful in the other.

In two instances division of aberrant vessels was thought to be sufficient treatment. Both pelves continue to have the same degree of dilatation but all pain has disappeared.

It is doubtful whether any of the last four procedures was truly the operation of choice.

RESULTS

Earlier attempts to classify patients according to the terms "well", "improved" and "failure" have been abandoned. All formulas for gradation of postoperative results have been inadequate, for none is flexible enough to compensate for the original status of the individual or for the indeterminate recuperative power of the affected kidney.

Although 42% of the patients now have normal pyelographic findings and no pain,

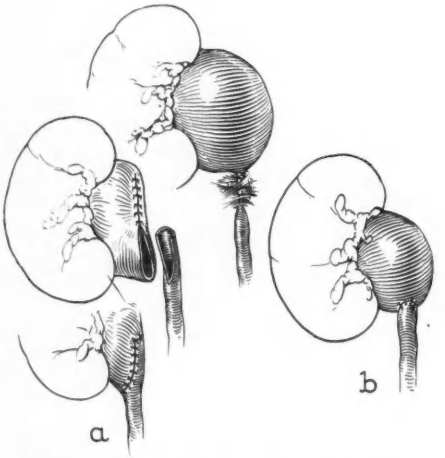


Fig. 4.—Type 4 obstruction. Postoperative scarring in ureteropelvic region; correction by ureteral reimplantation. (**a**) When possible, the spatulated end of the ureter is anastomosed with the pelvis over sufficient distance to produce a new funnel-shaped juncture. (**b**) When the renal pelvis is fixed and the ureter is short, only simple reattachment in the lower portion of the pelvis may be possible.

TABLE V.—RESULTS OF 223 PYELOURETEROPLASTIES

Type	*Total*	*Success*	*Failure*
Y-plasty.....	78	74	4 (5.1%)
Spiral flap.....	113	107	6 (5.3%)
Intubated ureterotomy...	16	16	0
Reimplantation..	9	7	2
Others.....	7	5	2
Total.....	223	209	14 (6.3%)

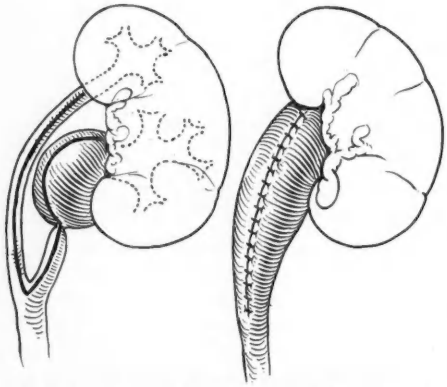


Fig. 5.—Ureteropelvic obstruction limited to the lower segment of a duplicated renal pelvis; correction by simple anastomosis of the pelves and corresponding segments of duplicated ureters.

infection or calculi, the most spectacula results were in individuals with kidney incapable of resuming normal size or con figuration because of permanent cortica damage.

For example, a man aged 37 years, wit advanced hydronephrosis, had a urea leve of 146 mg./100 c.c. of blood on admissio The level dropped to 34 mg. % afte pyeloureteroplasty. The solitary kidne now drains promptly but the pelvis still larger than normal and there periodic infection. The operation obviousl was successful, but the patient can hardl be considered entirely well.

One of the most significant confirmations of success has been the fact that 74 pa tients had calculi in the obstructed kidney and to date none has had a recurrence following pyeloureteroplasty at the time of pelviolithotomy.

Consequently, for practical purposes, the various operations have been classified simply as successes and failures despite the fact that the degree of success varied considerably (Table V).

Invariably, poor results were evident soon after operation. In successful cases, additional elapsed time revealed further improvement rather than belated deteriora tion. For example, a young Brazilian, aged 21 years, consistently had 500 c.c. of resi dual urine in her solitary kidney after Y-plasty and was assumed to have a failure. After one year of continued nephrostomy drainage, the renal pelvis emptied promptly and completely. She now is free of tubes, symptoms and infection, and has a gratify ing result.

As shown in Table V, there were 1 failures. Some of these probably would have been avoided if today's methods and standards had been employed.

Two of these kidneys were salvaged by additional plastic procedures. It is regret table that the successful techniques were not used initially. Perhaps pyeloureteroplasty should have been tried again in some of the other 12 failures, but it did not seem warranted at the time.

Permanent nephrostomy drainage was employed for four failures in solitary kidneys. These had presented complicated

situations with little hope of success when the patients were first seen.

Nephrectomy followed eight of the failures. Three of these represented kidneys that should have been removed at the initial operation. But the other five failures occurred under seemingly ideal circumstances.

All failures, especially the inexplicable ones, have been studied in minute detail in hope of finding a responsible and controllable common denominator. Severe and extensive ureteritis and periureteritis had produced ureteral stenosis in each patient who underwent a subsequent exploratory operation. All of these ureters had been splinted but none of the disastrous postoperative ureteral occlusions occurred at the end of a splint. One is reluctant to incriminate the tubes alone when they seemingly enhanced rather than compromised the results in more than 90% of the operations.

Perhaps it is significant that no failures have occurred after the last 103 pyeloureteroplasties. Probably it is safest to conclude that the healing properties of some ureters are unpredictable, regardless of surgical technique or experience.

Adjuncts

Of late there has been increasing enthusiasm throughout the literature for abandoning ureteral splints and diversion of urine. The desirability and results of such innovations cannot be questioned, but the wisdom of abruptly discarding all tubes in all cases must be viewed with skepticism.

Splinting and diversion of urine were omitted in 20 fairly recent cases. These patients were selected solely on the basis of the calibre of ureter with which the flap of pelvis was to be anastomosed. Some were infected. A few had calculi. Most had a localized bulbous dilatation just below the obstruction that permitted accurate approximation of tissues (Figs. 6(a) and (a')), in contrast to the difficulties encountered when only a thin ribbon of ureteral tissue was available for considerable distance (Figs. 6(b) and (b')).

To date there have been no failures. Although this small group lacks statistical significance, a few pertinent facts became self-evident. When all goes well there is no question about the reduction in postoperative morbidity despite the impression that pelves and calyces return to normal more slowly. One patient never leaked urine, had no infection and left the hospital within one week.

The duration of urinary drainage is unpredictable despite meticulous suturing. In one case it persisted for 28 days. The average for the group was 12 days. A constant urinary poultice can be ignored more easily by the surgeon than by the patient. It is annoying and uneconomical and demands continued hospitalization. Indwelling ureteral catheters might have reduced this morbidity but they automatically would have defeated the primary purpose of the tubeless technique. Most patients with splints and nephrostomy tubes left the hospital on or about the seventh postoperative day, even though they had infected urine. Admittedly, the time necessary to eliminate this infection after removal of the tubes was unpredictable.

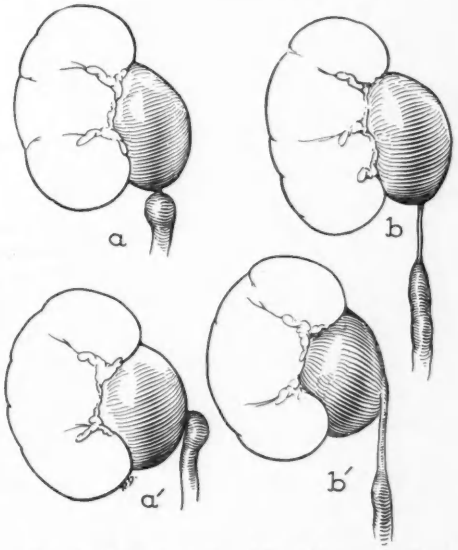


Fig. 6.—Variations in calibre of upper end of the ureter. Bulbous dilatations associated with **(a)** dependent juncture and **(a')** high ureteral insertion, both types of which are amenable to techniques devoid of splints. Narrow segments of ureter with **(b)** dependent juncture and **(b')** high ureteral insertion which require some type of splinting.

Delayed intrarenal bleeding can be especially harassing. One patient whose tissues had healed and who was apparently ready to leave the hospital on the eighth postoperative day failed to show any function in the kidney operated upon, on excretory urography. Retrograde pyelography revealed a pelvis filled with blood clot. A nephrostomy tube would have been welcomed at this point. Indeed, it might have prevented this predicament. After two weeks of lavage with streptokinase-streptodornase (Varidase) through indwelling ureteral catheters, the pelvis became free of the clot. Fortunately, the kidney appeared normal 12 months later.

When there is only a narrow strip of ureter to anastomose to the flap of pelvic wall and the two parallel rows of sutures must be so close to each other that viability of the intervening tissue may be jeopardized (Figs. 6(a) and (b')), splinting seems to be desirable. Soft rubber splints have been much more satisfactory than plastic ones. The calibre has varied considerably but sizes 12 F. to 16 F. have been used most frequently. An extra aperture is made in the intrapelvic portion of the splint. This is a welcomed auxiliary outlet for the urine if the nephrostomy tube becomes dislodged or obstructed.

With Y-plasties and spiral-flap operations a urethral catheter of appropriate size is passed down the ureter a reasonable distance and left in place for two weeks. With intubated ureterotomy and reimplantation a Levin tube is used to splint the *entire* ureter for four to eight weeks. All splints are brought out adjacent to a nephrostomy tube, which usually is a 22 F. urethral catheter. The latter is removed 24 hours after removal of the splint, provided clamping of the nephrostomy tube is followed by satisfactory emptying of the renal pelvis.

Aberrant renal vessels are usually preserved. Chemotherapy and antibiotic therapy are seldom given prophylactically, but most patients receive one or more antibacterial agents after all tubes have been removed.

Just as splinting unquestionably entails certain hazards, so does absence of splinting and of urinary diversion. While it s obvious that tubes are not necessary n all cases, selection of appropriate can - dates for tubeless methods continues to e a major challenge.

Other Complications

A 77-year-old man died of pulmona y embolism seven days after nephrecton . Except for the 14 failures that follow d plastic procedures, and the varying degre s of urinary infection that accompani d splinting and diverting tubes, these o - erations were followed by astonishing y few complications, as shown in Table VI.

Summary

Satisfactory treatment of ureteropelvic obstructions can tax the ingenuity of the most experienced surgeons. Since no one has infallible surgical judgment, even the choice between nephrectomy, nephrostomy and pyeloureteroplasty will require considerable deliberation in many instances.

After 411 operations on 385 patients with troublesome degrees of ureteropelvic obstruction, it became apparent that nephrectomy is indicated in a large percentage of cases, but the decision to remove a kidney cannot be based solely on its functional status.

Preliminary nephrostomy alone should be confined to rare situations in which concomitant correction of the obstruction might be unduly hazardous.

No one type of plastic operation will suffice for all varieties of obstruction. In this study, Y-plasty and the spiral-flap procedure were found to be of comparable value when adapted to properly selected cases. Intubated ureterotomy has earned a permanent role in the management of specific types of obstruction but has worked best when combined with Y-plasty.

TABLE VI.—Extraurinary Complications After 411 Operations

Type	*Number*
Fatal pulmonary embolism	1 (0.2%)
Phlebothrombosis	2
Excessive bleeding	3
Severe wound infection	3
Transient septicæmia	2
Total	11 (2.7%)

or the spiral-flap operation. Reimplantation has not been a very dependable solution to the complicated postoperative stenosis but these vexing problems are difficult to correct by any method. The legitimate role of ureteral splints remains controversial.

In all probability, newer and even better techniques will evolve, but one must be careful not to become a faddist. The ureteropelvic juncture always has been and no doubt always will be treacherous ground that should command everyone's respect. Injudicious selection of patients and poor choice of operations are a combination that will pay off only with grief and disappointment.

Résumé

Lorsque la fonction rénale est excellente, que les calices sont conservés et que le malade ne présente pas de symptômes, de pyurie ou de calculs, il est imprudent de "trifouiller" avec une obstruction urétéro-pelvienne. Plusieurs patients ont été revus annuellement depuis plus de 10 ans sans qu'on puisse observer de changement résultant de leur obstruction rénale relative. D'un autre côté, diverses associations de douleurs, d'infection, de calculs et de pyélocaliectasie progressive motivent l'intervention chirurgicale.

Le traitement adéquat de l'obstruction urétéropelvienne peut éprouver l'ingéniosité des chirurgiens les plus expérimentés. Puisque personne ne possède de jugement chirurgical infaillible, il nous faudra souvent délibérer soigneusement avant de choisir entre la néphrectomie, la néphrostomie ou la pyélourétéroplastie.

L'expérience acquise au cours de 411 opérations pratiquées sur 385 malades souffrant d'obstruction urétéro-pelvienne symptomatique, a démontré que la néphrectomie était indiquée dans un grand nombre de cas, quoique la décision finale ne puisse reposer seulement sur l'état fonctionnel du rein.

La néphrostomie préliminaire devrait être réservée aux rares situations où la correction concomitante de l'obstruction serait trop hasardeuse.

Aucune technique plastique isolée ne peut corriger toutes les formes d'obstruction. Dans cette étude, la plastie en Y et le lambeau en spirale furent également utilisés et requis selon le cas, L'urétérostomie par intubation s'est assurée une fonction permanente pour traiter certains genres d'obstruction mais a surtout été efficace lorsqu'associé à l'une ou l'autre des opérations. La réimplantation a pauvrement résolu le problème de la sténose post-opératoire mais ces complications ennuyeuses sont de correction difficile de toute façon. Le rôle légitime des supports urétéraux reste controversable.

En toute probabilité, il se créera des techniques nouvelles et meilleures, mais attention: la jonction urétéro-pelvienne a toujours été et sera sans doute toujours un terrain redoutable. La sélection malavisée des malades et le choix limité des opérations constituent une combinaison qui résultera en peine et déception.

SURGERY OF THE HAND IN CEREBRAL PALSY AND THE SWAN-NECK DEFORMITY*

"Any surgical procedure to improve function of the hand in cerebral palsy must be regarded as a compromise because normal function can never be attained. The disabilities of the hand in this disease often present therapeutic problems that are more complicated than those encountered in traumatic and paralytic deformities. These problems are complex because the functional equilibrium that exists between the intrinsic and extrinsic muscles of the normal hand is often disturbed by muscle spasm or paresis and the associated deformities and contractures that characterize cerebral palsy. Furthermore, the normal functions of contiguous articulations in the fingers and hand are interdependent and any disturbance in one joint will affect other, related joints. In cerebral palsy these interrelated functions are profoundly affected and it may be difficult to determine which deformity is primary and which is secondary.

"The majority of deformities of the upper extremity in cerebral palsy are best treated by physical measures such as bracing, stretching, and muscle reeducation. In a selected group of cases, certain operative procedures, if done when properly indicated, may assist in the patient's rehabilitation and enable him to perform daily tasks more efficiently because the disabled hand, although not made normal, can now be trained as an efficient assistive member.

"The major handicap of the hand in cerebral palsy is the patient's inability to open the hand for grasp. Often, the hand has good strength for closing but does not have sufficient extension or the necessary speed of extension, which is so important for normal function. . . ."

*Swanson, A. B.: *J. Bone & Joint Surg.*, **42-A**: 951, 1960.

WEIGHT-BEARING BY LIGAMENTS AND MUSCLES*

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HARDLY ANY informed person would doubt that when gravity acts on the upper limb, and certainly when the limb carries a heavy load, muscles are the chief agents in preventing the distraction of the joints. Yet, recently, I have been forced to conclude that in the absence of major qualifications this is a false belief. Having originally stumbled on this idea by accident while working on the electromyography of shoulder muscles with Dr. F. J. Bazant,[2] I extended our observations further to include the elbow region. Essentially, the fundamental conclusion can be made that ligaments play a much greater part in supporting loads than is generally thought and, in most situations where traction is exerted across a joint, muscles play only a secondary role. A review of our experiments on the foot (reported in the past with J. W. Bentzon[3]) adds further confirmation to the idea that, normally, ligaments and not muscles maintain the integrity of joints.

MATERIAL AND METHODS

GROUP 1:—SHOULDER AND ELBOW REGION

Our study in this region has already been reported in detail.[2] The electromyographic investigation dealt with supraspinatus, infraspinatus, deltoid, biceps and triceps muscles in a series of 22 normal persons using multiple bipolar needle electrodes. In eight subjects the three parts of the deltoid (anterior, middle, and posterior) and the supraspinatus were studied simultaneously (in one of these subjects, the biceps was also studied simultaneously). In 11 subjects, the supraspinatus and infraspinatus were studied simultaneously. In five subjects, the biceps was studied; in four of these the long head of the triceps was studied simultaneously.

The subjects were all examined in a comfortable, seated position. At each site chosen for insertion of an electrode a small skin wheal was raised with 1% procaine and through this the electrode was thrust into the muscle. In the case of the deltoid, the needle in the anterior fibres was two inches below the lateral end of the clavicle; that in the middle fibres was two inches below the lateral border of the acromion; and that in the posterior fibres was about three inches below the spine of the scapula. The electrodes in the supraspinatus and the infraspinatus were placed in or near the middle of their bellies. The electrodes in the biceps were placed in the middle of the muscle whereas those in the triceps were placed in the middle of its long head. In no instance was pain experienced by the subjects during these experiments (Fig. 1).

The electrodes used were concentric-needle electrodes of a type described previously.[1] The electromyograph is a special six-channel apparatus consisting of six separate amplifiers and cathode-ray oscilloscopes whose faces can be photographed simultaneously on the same strip of continuous-moving 35 mm. linagraph film.[1] Even the slightest electromyographic activity can be accurately recorded (Figs. 2 and 3).

Although several tests other than those to be described were performed (for example, various movements of the shoulder joint), only those concerning the present inquiry will be considered here.

A. Static unloaded hanging arm: The subject was seated upright with his arm hanging in the relaxed neutral position (the forearm midway between pronation and supination).

B. Static loaded hanging arm: Two types of load were added to the subject's arm. The first of these was a load of 16 lb. (lead weights held in the hand to the limit of individual endurance which proved to be a variable factor). The other load, less precise but more effective, was a sudden heavy sustained downward pull by one of the observers on the subject's hanging arm.

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*From the Department of Anatomy, Queen's University, Kingston, Ontario. This work was supported in part by grants from the National Research Council of Canada and the Muscular Dystrophy Association of Canada.

†Professor and Head of the Department of Anatomy, Queen's University, Kingston, Ontario.

This paper was submitted for publication in June 1960.

In five persons a longitudinal pull was applied to the arm which had been abducted to the horizontal plane and completely supported by another observer so that no abduction activity was required of the subject's muscles.

Group 2: Elbow Region

Twenty-four young adults were studied electromyographically with multiple needle electrodes in both heads of the biceps, in the brachialis, and in the brachioradialis. Although many studies of the patterns of movement were performed and reported for a smaller number of subjects elsewhere,[4] the present study concerns only the static hanging forearm, with and without added loads. In addition, the pronator teres muscles of eight other subjects have been studied more recently (with the assistance of Dr. A. Travill).

In all instances, electromyographs were made with the subject seated upright and the upper limb hanging straight downwards in a comfortable position. In Group 2, when the main flexors of the elbow were studied, the added load was a weight of 2 lb. Thus considerable numbers of biceps muscles were studied, some with heavy and moderate loads in Group 1 and others with light loads in Group 2. In making the electromyograms of the pronator teres in Group 2, only the strong downward pull was used as the added load, since experience had already shown the ineffectiveness of lesser loads.

Observations

Shoulder Region

A. Static unloaded hanging arm: In this position electromyographic activity in all three parts of the deltoid ceased almost immediately. Meanwhile, the activity in the supraspinatus fell off and eventually reached nil. However, the length of time between electrode insertion and nil activity of the supraspinatus varied from immediately in several subjects to one hour in one subject. The infraspinatus, like the deltoid, relaxed completely almost immediately in all subjects, while the biceps and the triceps showed no activity, either intermittent or continuous (Figs. 4 and 5).

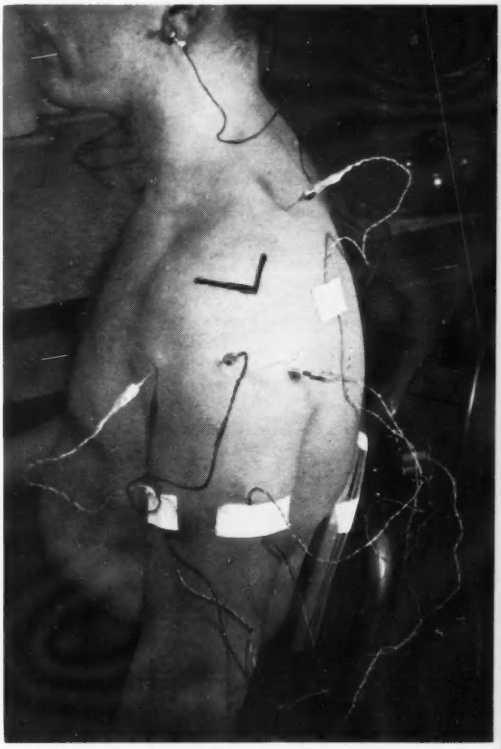


Fig. 1.—A subject in experimental position with needle electrodes in three parts of deltoid and in supraspinatus.

B. Static loaded hanging arm: The limit of endurance in the subjects holding the 16 lb. load varied from three to 13 minutes. (Weight-holding was discontinued in each person because of the fatigue experienced in the forearm and hand in maintaining the unchanging and, therefore, uncomfortable position.) With this load, the supraspinatus varied from subject to subject, ranging from no electromyographic activity to marked activity, being quite active in general. The posterior (horizontal) fibres of the deltoid showed a range from no activity to only moderate activity. The anterior and middle fibres, in contrast, showed no activity in almost all the subjects and in the two subjects in whom there was some activity, it was quite slight. Moreover, when activity was present, there was some slight rise and fall from minute to minute.

During the sudden but sustained heavy

pull downward, the surprising result was that there was no appreciable increase in activity of the anterior and middle fibres of the deltoid. However, the activity in the supraspinatus and the posterior (horizontal) fibres of the deltoid increased appreciably in all but one subject during the downward pull applied to the arm. In the case of the horizontal pull, the results were similar; again the activity of the anterior and middle fibres of the deltoid muscle was not increased, whereas the activity in the posterior deltoid and supraspinatus was increased.

There was no activity in the biceps and triceps muscles even when the arm was pulled vigorously downward (Fig. 5).

Elbow Region

In all of the 24 concurrent studies of the biceps, brachialis, brachioradialis and triceps and in all of the eight studies of the pronator teres, activity was entirely absent from these muscles in the relaxed hanging position of the limb.

The muscles remained quite inactive in all of these subjects when the load was added, whether it was the light load in the former group or the heavy load in the latter.

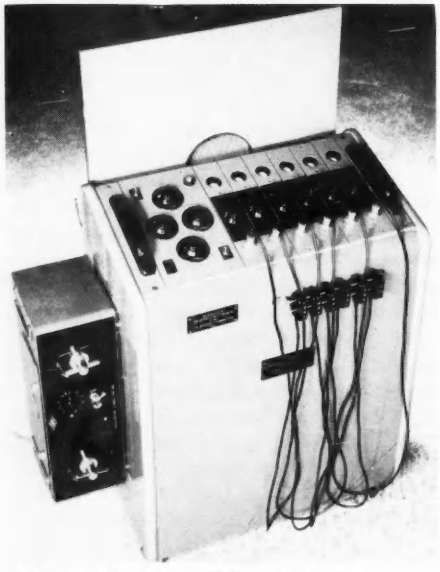


Fig. 2.—Special multichannel electromyograph built for the author by Stanley Cox, Ltd. Note on upper surface, six amplifiers each with its own small cathode ray tube for monitoring. Linagraph camera (on left side) makes continuous recordings from another set of c.r. tubes within cabinet.

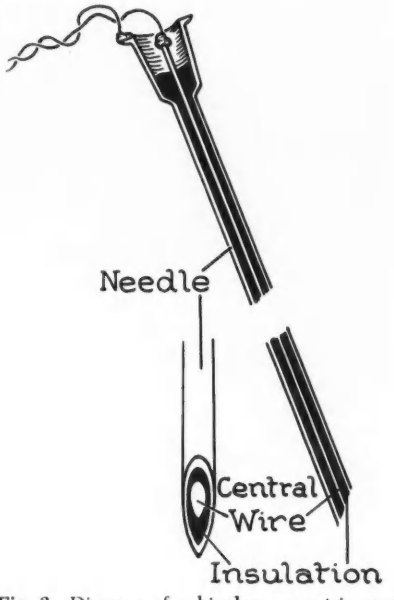


Fig. 3.—Diagram of a bipolar concentric needle electrode.

Discussion

Contrary to expectation, the vertically running muscles that cross the shoulder joint and the elbow joint are not active to prevent distraction of these joints by gravity. Much more surprising is the fact that they do not spring into action when light, moderate, or even heavy loads are added unless the subject voluntarily decides to flex his shoulder or his elbow and thus to support the weight in bent positions of these joints. Quite often, he may do this intermittently or, when unistructed, from the very onset. But it must be clear that such muscular action is voluntary, not a reflex action.

Even while the muscles were quiescent, our subjects rapidly felt local fatigue. What then, is fatigue in the heavily loaded limb? Normally, it would be thought of as "muscular fatigue" but we see now that this is patently incorrect. The "fatigue" that is experienced probably originates from

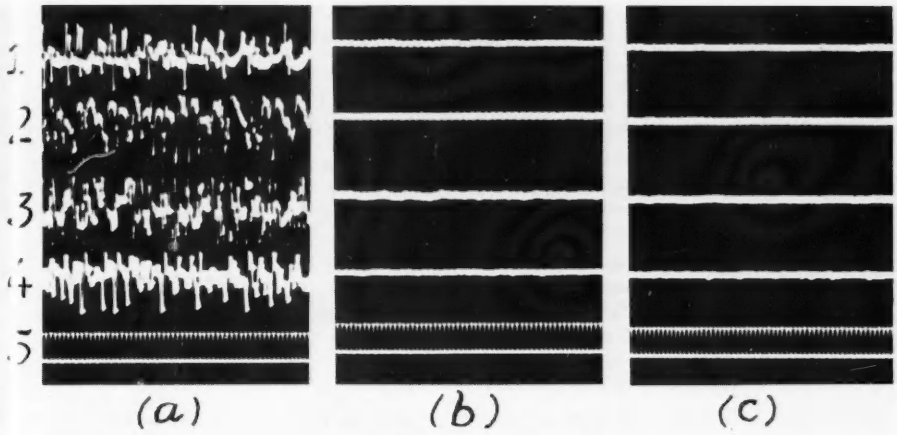


Fig. 4.—Electromyograms of three parts of deltoid and of supraspinatus, (a) during abduction, (b) with unloaded arm hanging and (c) with heavy load on hanging arm. Lines 1, 2 and 3 are records of anterior, middle and posterior parts of deltoid; 4 is supraspinatus; 5 is calibration signal, frequency 100 c.p.s., amplitude 200 microvolts. Note the very slight activity in horizontal muscles and total silence in vertical ones.

the painful feeling of tension in the articular capsule and ligaments, not from overworked muscles. In fact, as we have seen, the muscles need not be working at all.

An analogous situation occurs in the foot where we found, some years ago, that the muscles that are usually supposed to support the arches continuously were generally inactive in standing at rest.[3] Independently, Hicks[5] showed by deduction that the plantar aponeurosis and plantar ligaments were the chief weight-bearers in this position. It would seem, then, that in the normal foot the fatigue of standing is not a muscular phenomenon.

The dual conclusion that articular ligaments suffice to prevent the downward distraction of joints in the upper limb, and that fatigue is chiefly a form of pain in the ligaments, appears to me to be of fundamental importance. It not only runs counter to "common sense" but it is of practical interest, for example, in explaining why dislocations by traction on normal limbs are rare. It should be noted especially that the capsule on the superior part of the shoulder joint including the coracoacromial ligament is extremely tight only when the arm hangs directly downward and the scapula is in its normal position. The special mechanism that includes this ligament together with the supraspinatus muscle and the normal slope of the glenoid cavity, have been described elsewhere.[2] When the shoulder joint is abducted or flexed, however, the capsule is extremely loose and

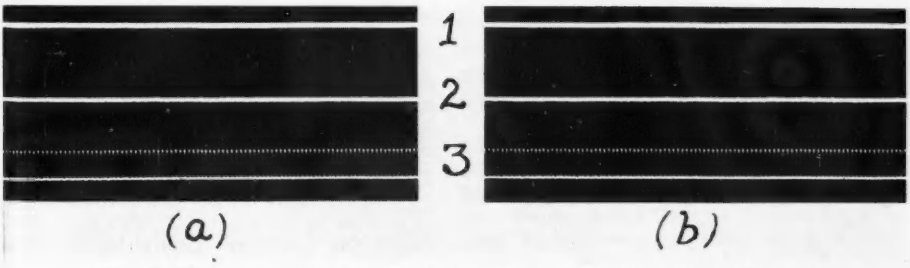


Fig. 5.—Electromyogram of biceps (line 1) and triceps (2): (a) unloaded hanging limb, and (b) heavily loaded. There is no change. (Calibration signal on line 3 has frequency of 100 c.p.s. and amplitude of 200 microvolts.)

the shoulder joint depends for its integrity on the well-known "rotator-cuff" muscles.

Summary

Forceful distraction of the elbow joint is prevented chiefly by ligamentous structures rather than by muscles. The same is true of the shoulder joint only in the vertical (adducted) position of the arm. These concepts emerged from a long series of electromyographic studies using needle electrodes in the supraspinatus, infraspinatus, deltoid, biceps, triceps, brachialis, brachioradialis and pronator teres. Analogously, the ligaments of the foot and not the muscles, form the chief defence against flat feet. The resultant "fatigue" in heavily loaded limbs in the absence of even mild exertion by muscles is actually a form of pain in the ligaments and not in the muscles.

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Résumé

L'arrachement violent du coude est surtout empêché par l'architecture ligamentaire plutôt que par les muscles. Il en est de même pour l'épaule, mais seulement en position verticale (en adduction) du bras.

Ces concepts dérivent d'une longue série d'études électromyographiques par aiguilles polarisées sur le sus-épineux, le sous-épineux, le deltoïde, le biceps, le triceps et les groupes de l'avant-bras. De façon analogue, les ligaments du pied et non les muscles constituent la principale protection contre les pieds plats.

La fatigue qui résulte du chargement lourd des membres en l'absence du moindre effort musculaire est en fait une forme de douleur au niveau des ligaments et non des muscles.

SOCIALIZED MEDICINE IN SWEDEN*

"The Scandinavian countries usually are regarded as having the most efficient socialized medical program. Sweden is to spend the equivalent of $50,000,000 to bring within the reach of all inhabitants special care for patients with heart disease, cancer, nervous disorders, and other ailments. By increasing their facilities and adding 250 physicians and 500 nurses, there will be no waiting and no need to travel far in order to get the best possible treatment.

"Sweden has had compulsory health insurance since 1955. The people receive free care in a hospital ward, substantial refunds on private doctors' bills and prescriptions, payment of daily sickness benefits, and free maternity care. They have free choice of physicians, and according to release No. 799 from the American-Swedish News Exchange, private practitioners may charge any fee they wish. They are reimbursed by the sickness funds according to certain standard rates.

"It is not as free as it sounds. One-half the cost is covered by individual premium payments, one-fourth by the employers, and one-fourth by the government, that is, the taxpayer. The annual premium for a person living in Stockholm is approximately $8, and another $6 is paid for the basic sickness cash benefits. Children are covered at no extra cost.

"Sweden has fewer physicians and surgeons in relation to the population than the United States, but no other country has so many nurses and auxiliary hospital personnel. The number of hospital beds is higher than in most industrial nations."

*Editorial, *Illinois M. J.*, **118:** 109, 1960.

RETICULUM CELL SARCOMA OF THE TESTIS: REPORT OF TWO CASES

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PRIMARY lymphomatous tumours of the testes are rare. At the Mayo Clinic, Dockerty and Priestley[1] encountered only four in 500 tumours of the testis. Cohen *et al.*[2] reviewed 2860 cases of testicular tumour in the English language literature published between 1945 and 1955. They could find only six cases of reticulum cell sarcoma primary in the testis, although this organ was a fairly common site of secondary tumours, especially in the patient over 60 years of age. They reported four more cases of reticulum cell sarcoma from their own experience of 233 primary testicular tumours. Mathe[3] found one case of lymphosarcoma in a series of 34 patients with primary testicular malignancies. Hotchkiss and Laury[4] reported a case of bilateral reticulum cell sarcoma of the testis in a man 67 years of age and reviewed 21 more cases of bilateral testicular tumours reported in the literature. About one-third of these were lymphoblastomas. Cohen *et al.*,[2] Hotchkiss,[4] Symmers,[5] Fabian[6] and Ewing,[8] have stated that lymphomatous tumours are quite often bilateral, but Hamilton and Gilbert[7] in a search of 7000 case records, found only 134 cases in which both testes were involved with cancer other than lymphosarcoma. Indeed, they suggested that a man who has had a carcinoma in one testis is much more likely to develop carcinoma in the other than a normal individual. Twenty-one cases of concomitant bilateral testicular malignancy have been catalogued in the literature, and a large number of these are lymphosarcomas.

In the Toronto Western Hospital in the last 20 years, the Department of Pathology has recorded two cases of reticulum cell sarcoma in the testes among 52 testicular tumours examined during that period. One of these was bilateral. The only other bilateral tumour was embryonal carcinoma in a man with both testes undescended.

*†Toronto Western Hospital.

Case Reports

Case 1.—An 80-year-old Russian was first admitted to the Toronto Western Hospital on March 23, 1958, with a history of painless swelling in the left testis of five months' duration. There had been no loss of weight, appetite or vitality. No other masses had been noted. Functional enquiry and history of past illnesses were non-contributory.

Examination.—The left side of the scrotum was occupied by a mass replacing the testis, measuring three and one-half by two inches by two inches. It was oval, firm, smooth, non-tender, freely movable and not attached to the skin. The epididymis could not be palpated separately from the mass. Signs of mild congestive heart failure and prostatic hypertrophy were present. Hæmoglobin was 92% and the white blood cell count was 6700/c.mm. The differential white blood count showed 47% neutrophils, 34% lymphocytes, 7% eosinophils, 1% basophils and 11% monocytes, with no circulating reticulum sarcoma cells. The Ascheim-Zondek test was negative and the non-protein nitrogen was 39 mg. %. The urine had a specific gravity of 1.018 and was negative for albumin, sugar and acetone. Microscopic examination was negative.

Treatment.—Orchidectomy was performed on March 24, 1958. The pathological report was "reticulum cell sarcoma of the testis" (Fig. 1). Direct invasion of the spermatic cord to within one inch of the level of cord division had occurred. Because no evidence of generalized disease could be found, he was discharged from hospital following an uneventful recovery from the operation.

This patient was admitted for the second time on May 22, 1958. This readmission was occasioned by the development of bilateral swellings in the neck, a weight loss of 20 lb., malaise, loss of vitality and vague abdominal pain.

Examination.—There was enlargement of lymph nodes in the anterior and posterior cervical groups and submandibular groups bilaterally. The axillary and inguinal nodes were enlarged, firm, smooth, rubbery, discrete, movable and non-tender. The spleen was not palpable but was thought to be enlarged to percussion, and the mediastinum was widened to percussion. Glycosuria was present, and in-

vestigation revealed mild diabetes which was suspected to be due to pancreatic infiltration. It was easily controlled with insulin and dietary regimen. Hæmoglobin was 14.0 g. % and leukocytes were 6500/c.mm., with a normal differential count and no circulating reticulum cell sarcoma cells.

Treatment.—The patient was treated with nitrogen mustard (methyl-bis-betachlor-ethylamine hydrochloride) 1 mg./kg. of body weight daily for four days, and with Duracton, 40 units intramuscularly daily. There was a marked response as shown by a rapid reduction in the size of the involved lymph nodes, but the malaise persisted. He was discharged from hospital to be followed up in clinic.

Course.—Within one week the superficial lymph nodes rapidly enlarged. He went downhill and died four weeks after cessation of the nitrogen mustard therapy. Permission for postmortem was not granted.

CASE 2.—In August 1957 a 39-year-old German presented with swelling of the left testis and intermittent pain of three months' duration. Functional enquiry revealed no loss of weight or deterioration of his general health. The history of past illnesses and his occupational history were non-contributory.

Examination. — General physical examination was normal. The left testis was enlarged to twice the normal size, and contained a mass which was smooth, firm and non-tender. The epididymis was not involved. The skin overlying the testis anteriorly was slightly reddened.

Hæmoglobin was 13.4 g. %, erythrocytes were 4,600,000/c.mm., and the white blood count was 7200/c.mm.

Treatment.—Left orchidectomy was performed on October 15, 1957. Involved inguinal skin was removed along with the testis and cord. Convalescence was uneventful. A pathological diagnosis of seminoma was made and he was treated with radiation therapy. He received 3000 roentgen units (air dose) to the abdominal para-aortic lymph nodes, left groin and spermatic cord. He presented for readmission to hospital five months later, in January 1958, with a painless swelling of the right testis but with no deterioration in general health and no weight loss.

Examination.—The right testis was now enlarged and replaced by a tumour which was smooth, rubbery and non-tender.

Treatment.—Right orchidectomy was carried out on January 29, 1958. A pathological diagnosis of reticulum cell sarcoma was made (Fig. 1). Review of the sections of the left-sided tumour resulted in revision of the original diagnosis to reticulum cell sarcoma.

Course.—Within a short time, nodules appeared in the skin of the trunk. Abdominal swelling, loss of weight, anorexia, malaise and loss of vitality rapidly developed. Examination revealed a tumour mass within the abdomen and an intravenous pyelogram showed that this mass caused lateral displacement of the upper ureters. Large, hard, discrete, movable lymph nodes developed in the right axilla, in the supraclavicular and posterior cervical chains and in the right inguinal region. Hæmoglobin was 8.7 g. %. The leukocyte count was 2900/c.mm. Biopsy of a skin nodule was performed and the pathological diagnosis was cutaneous lymphoma.

High voltage therapy to the abdomen, left axilla and the left neck failed to influence the lymph node enlargement. Treatment with one dose of nitrogen mustard, 25 mg. intravenously was given. For a brief period there was dramatic shrinkage in size of all superficial lymph nodes and the abdominal mass. Within 10 days re-enlargement of all the involved nodes was noted. He went downhill rapidly and died of generalized reticulum cell sarcoma six weeks following nitrogen mustard therapy.

DISCUSSION

Gall and Mallory[10] in reviewing 194 cases of lymphoma in 1942, stated that the relative frequency of the various primary sites depended on the bulk of lymphoid tissue in the different regions of the body. There is so little reticuloendothelial tissue in the testis, and it is said to be functionally so dormant, that it is rare for reticulum cell sarcoma to arise in this organ. In fact, Chevassu[9] has stated that all lymphosarcomas in the testes are secondary to lymphosarcoma elsewhere. However, the weight of pathological opinion seems to be in favour of the view that it can arise primarily in the testis. According to Maximow,[11] the interstitial collagenous tissue of the testes contains a network of blood and lymph vessels, some lymphocytes, monocytes, fibroblasts, fixed macrophages, mast cells, embryonic perivascular elements and undifferentiated mesenchymal cells. It is probable that the malignant transition to reticulum cell sarcoma takes place in the undifferentiated mesenchymal cells. This is followed by compression of the seminiferous tubules by the diffuse

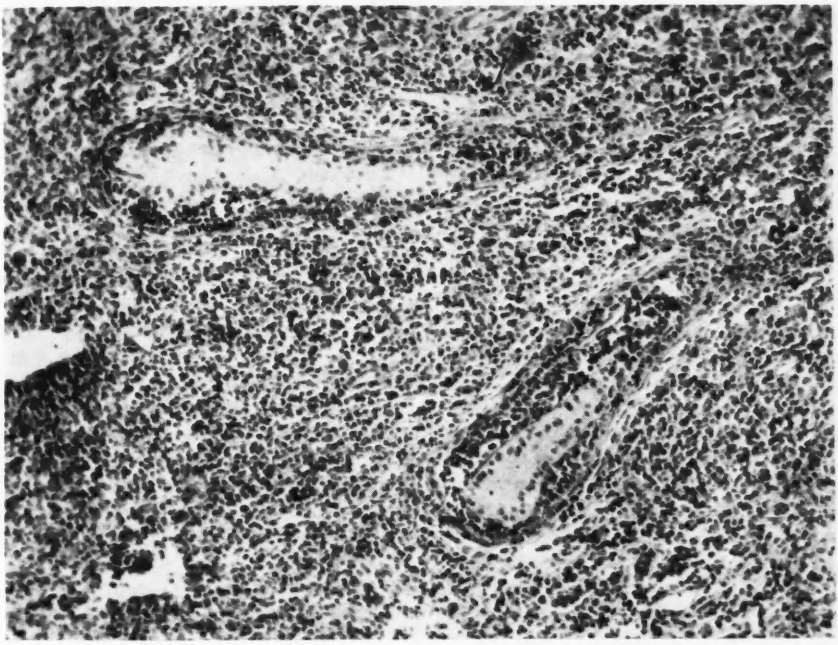


Fig. 1.—Section of the left testis of Case 2. This section is representative of all three reported tumours. Sheets of tumour cells largely replace the testicular tissue. The tumour cells infiltrate the seminiferous tubules and lift the basement membrane of the germinal epithelium from the supporting connective tissue stroma. Special stains demonstrate a moderate increase in reticulin fibrils (H & E. x 80).

invading sheets of cells, invasion of lymphatics with spread to the regional lymph nodes, and hæmatogenous spread to distant organs such as the lungs, heart, tonsils, suprarenal glands and bone. Involvement of lymph nodes at distant sites occurred in both of our patients.

Because seminoma is much more common, and because reticulum cell sarcoma of the testis frequently behaves like seminoma, one is inclined to mistake the one for the other. This occurred in Case 2, in which the original pathological diagnosis was seminoma of the left testis. It was not until the patient was admitted to hospital five months later with swelling of the right testis that the diagnosis of reticulum cell sarcoma in both testes was made. The pathologist described a homogeneous grey-white tumour mass replacing and invading the testicular tissue, but limited by the thin capsule around the testis. The tumour was composed of pleomorphic cells with little or no cytoplasm and large irregular nuclei. Moderate numbers of mitotic figures were present. The seminiferous tubules were compressed and atrophic. This picture is similar to that described in the literature.[2, 8, 13, 16, 17] Cohen *et al.*[2] put considerable diagnostic emphasis upon the observation that reticulum cell sarcoma does not invade the seminiferous tubules, but grows between the tunica propria causing compression and atrophy of the tubules, only penetrating at a few sites where the tubules were completely necrotic, whereas the tunica propria does not seem to be a barrier to seminoma cells and they are often seen within the tubules. Herbut,[16] on the other hand, states that the tubules are permeated with lymphoblastoma cells as they undergo degeneration and atrophy due to encroachment. The two cases reported in this communication had tumour cells infiltrating the seminiferous tubules and lifting the basement membrane (Fig. 1). Special stains demonstrated an increase in reticulin fibrils and

helped to identify the tumours as reticulum cell sarcoma rather than seminoma.

Treatment consists primarily of orchidectomy with or without postoperative irradiation of adjacent lymph nodes. Where the disease has become widespread, nitrogen mustard therapy is occasionally of some value. We chose to give the first patient 0.1 mg./kg. daily for four days, and the second patient 25 mg. in one injection. Both patients were also given 40 units of repository ACTH daily for 24 to 48 hours before the nitrogen mustard, and during the course of treatment. This seems to reduce the adverse side effects of nitrogen mustard therapy. It also serves to reduce the possible antigenic effect of a sudden flood of reticulum cell protein into the circulation. This might stimulate an antigen-antibody response, and the appearance of acquired hæmolytic anæmia due to red cell coating antibodies. Further, there is some evidence that patients who fail to respond to either the alkylating agents or corticosteroids alone may respond when both these agents are used together, and it is possible that ACTH or cortisone therapy will allow a greater dose of nitrogen mustard to be given without depression of the bone marrow. Chlorpromazine or cyclizine hydrochloride were used to combat nausea caused by the treatment. Larger amounts of nitrogen mustard have been used to treat other types of neoplasms such as metastatic carcinoma of the breast, lung or ovary, but it has been our experience that this is not without danger in the lymphomatous diseases. The bone marrow seems to be much more readily depressed by the chemotherapeutic agents when the patient suffers from lymphoma than when adenocarcinoma is present.

Treatment of these two patients was disappointing, since both had evidence of widespread metastases within a few months, and suffered a rapid downhill course. Both had a striking objective response to nitrogen mustard but it was short-lived, re-enlargement of lymph node metastases occurring within seven to 14 days of therapy.

The results obtained by Miller, Diamond and Craver[12] in the treatment of lymphosarcoma and reticulum cell sarcoma with chlorambucil (P-(di-2-chlorethylamino) phenylbutyric acid) are encouraging. This alkylating agent may prove to be more effective than nitrogen mustard.

Lawrence and Lenson[13] state that the life expectancy in reticulum cell sarcoma in any site is six to 24 months even with resection and irradiation, but Jenkinson *et al.*[14] report only a 20% recurrence in two years in those patients receiving 2000 to 3000 roentgens locally. Helwig[15] found that 24% lived five years after surgical extirpation of the primary tumour. Lawrence and Lenson[13] reported a patient living 13 years after irradiation, and some instances of 20 year survival have been recorded. Reticulum cell sarcoma of bone has a better than average prognosis, 50% of patients living five years. In contrast, our two patients with primary tumour in the testis, had a rapidly progressive course, death occurring within eight to 10 months of the first awareness of a testicular mass.

Summary

Two cases of reticulum cell sarcoma, primary in the testis, have been presented. The disease is uncommon in comparison with other types of testicular tumours.

Reticulum cell sarcoma of the testis is frequently bilateral.

The pathological picture is characteristic and it can be readily differentiated from seminoma.

Treatment consists of orchidectomy, followed later by local radiation. When multiple metastases appear, the alkylating agents may be of value.

Primary reticulum cell sarcoma of the testicle has a poor prognosis in contrast to reticulum cell sarcoma arising in some other sites.

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Résumé

Les tumeurs lymphomateuses primaires des testicules sont rares. A la clinique Mayo, Dockerty et Priestley[1] en ont rencontré seulement quatre sur 400 cas de tumeurs du testicule.

Au "Toronto Western Hospital", durant les 20 dernières années, le département de Pathologie a rapporté deux cas de réticulo-sarcome des testicules parmi 52 cas de tumeurs testiculaires examinés durant cette période.

Cette tumeur est souvent bilatérale et se distingue assez facilement du séminome, du point de vue histologique.

L'orchidectomie en constitue le traitement, suivie de radiothérapie locale. En présence de métastases, les agents alkylisants peuvent être utiles. Le réticulosarcome primaire du testicule a un pronostic sombre contrairement à celui qui se développe en d'autres sites.

BUTS DU TRANSPLANT*

"Le but du transplant que l'on poursuit en chirurgie biliaire ou pancréatique, lorsqu'on utilise un transplant, est double:

"1° Il s'agit parfois de ponter un canal, c'est-à-dire de remplacer une partie de ce tube par un autre tube, prélevé au niveau de l'intestin, sans cependant modifier ni le début ni la terminaison du canal. Cette méthode permet de sauvegarder l'appareil musculaire propre de la partie haute de la voie biliaire, par exemple (sphincter de Mirizzi), ainsi que de sa partie terminale (complexe oddien).

"2° Il faut, d'autres fois, remplacer un ensemble musculaire complexe sphinctérien, à activité propre extrêmement précise.

"Ce complexe, ainsi que nous l'avons montré dans les travaux antérieurs (180, 158), est animé de mouvements rythmiques, consécutifs à un péristaltisme orienté du canal cholédoque. Cette mobilité est d'ailleurs sous la dépendance de diverses excitations nerveuses, soit directes, soit réflexes, ainsi que de nombreuses excitations médicamenteuses ou hormonales.

"Il est bien certain que le remplacement d'un organe aussi complexe, par un transplant intestinal, ne sera jamais qu'un pis aller.

"C'est précisement en raison de la complexité du problème que de nombreuses solutions ont été préconisées.

"Le montage d'une anse jéjunale en Y semble, dans l'état actuel de nos connaissances, être l'artifice qui permet de se rapprocher le plus des conditions physiologiques normales."

*Stalport, J., Primo, G. et Gregoir, W.: L'utilisation des transplants intestinaux en chirurgie, *Acta chir. belg., Supp.*, **1:** 20, 1960.

THE CLINICAL USE OF POLYVINYL SPONGE (IVALON) IN THE REPAIR OF ŒSOPHAGEAL HIATUS HERNIA*

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THE PROBLEM OF œsophageal hiatus hernia has received increasing attention during recent years. This condition is demonstrated today with greater frequency by careful radiologists who are aware of the lesion, and it is also discovered by surgeons who palpate the œsophageal hiatus during a complete abdominal exploration. Despite many excellent reports concerning the repair of œsophageal hiatus hernia, including those of Harrington,[1] Allison[2] and Sweet[3] no surgical procedure has been uniformly adopted. The present study was undertaken to review certain anatomic and pathophysiological aspects of hiatal hernias, and to present a series of 17 patients in whom surgical treatment was performed utilizing Ivalon sponge as an adjunct in the repair.

ANATOMIC FEATURES

Several anatomic structures are significant; these are muscular, fascial, and peritoneal.[4] The œsophageal hiatus is a tear-shaped opening in the diaphragm produced in most instances by a splitting of the fibres of the right crus of the diaphragm (Fig. 1). The right crus commonly splits into a superficial layer forming the right margin of the hiatus, and a deep layer forming the left margin.[5] This crus forms a muscular sling which supports the œsophagogastric junction and maintains the normal relationship between œsophagus, diaphragm and stomach. Anteriorly the hiatus is strengthened by a decussation of muscular fibres and by the proximity of the central tendon of the diaphragm. The hiatus is relatively unprotected posteriorly where pressure will tend to enlarge the hiatus by splitting the crural muscle fibres along their longitudinal axis.

The phrenœsophageal ligament is that portion of the deep fascia which passes from the under surface of the diaphragm through the œsophageal hiatus and attaches to the fascia propria of the œsophagus about 3 cm. above the œsophagocardiac junction.[1] This ligament has received considerable attention in some descriptions of hernial repair.[1, 6, 7]

The peritoneal attachment at the œsophagocardiac junction is relatively loose anteriorly permitting invagination through the hiatus for several centimetres. This redundant tissue allows digital exploration of the hiatus by the surgeon at laparotomy and may form the source of a hernial sac.



Fig. 1.—Diagram illustrating diaphragmatic crura and œsophageal hiatus.

*Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Montreal, January 1960.

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Pathophysiology

The function of the œsophageal hiatus is to permit the passage of œsophageal contents into the stomach, and prevent reflux of gastric contents. The functional integrity of the hiatus is maintained by the oblique entry of the œsophagus into the stomach, an apparent œsophageal constrictor mechanism, and the muscular crural sling.

The slight angulation to the left of the distal œsophagus as it passes through the hiatus may act as a hinged valve mechanism to prevent regurgitation. Gastric distension increases the acuteness of the angle, and in effect, closes the valve. The oblique entry also protects against herniation. Increased intra-abdominal or intragastric pressure tends to force the cardia and fundus of the stomach against the diaphragm, rather than through the hiatus.

Although the presence of an intrinsic sphincter in the region of the œsophagocardiac junction is anatomically suspect, physiological studies have indicated the existence of a constrictor mechanism in the lower œsophagus just above the diaphragm.[8] This inferior œsophageal constrictor may act synergistically with the crural muscular sling to prevent gastric reflux.[9]

The crural sling encloses the lower œsophagus and maintains the size of the hiatal opening. It also provides active as well as passive resistance to dilatation of the hiatus. Active contraction of the muscular crus compresses the distal œsophagus, pulls it downward and narrows the oblique angle of entry into the stomach.

An œsophageal hiatus hernia is primarily a mechanical deformity and not a disease entity. The simplest classification is that of Allison.[2] Sliding hiatus hernia is the commonest form in which there is a direct herniation of the stomach through an enlarged hiatus with displacement of the œsophagocardiac junction above the diaphragm. A paraœsophageal hernia involves a protrusion of stomach through the hiatus alongside the œsophagus. The œsophagocardiac junction maintains a relatively normal position. A true hernial sac may or may not be present, and multiple variations and combinations of these hernial types are found.

The ætiology is indefinite in most patients with hiatal hernias, but predisposing factors include congenital anomalies, trauma, obesity, increased intra-abdominal pressure, and atrophy or disease affecting the anatomic supports of the hiatus. The incidence of this condition has been reported as high as 9% in a large series of patients who have had roentgenographic studies of the upper intestinal tract.[10]

Clinical Study

There were 12 women and five men in this series. All were over 38 years of age, and 13 were over 50 years. The oldest patient was 75. Obesity was present in either moderate or severe degree in 11 patients and a determined effort was made to have these patients reduce weight before surgical intervention.

Hiatus hernia has been termed "the great masquerader" of the upper abdomen and its symptoms may mimic those of gallbladder disease, peptic ulcer, pancreatitis, malignancy of the stomach or œsophagus and coronary artery disease. Differentiation is often difficult and complicated by the concomitant presence of these conditions, particularly gallbladder and coronary artery disease. A history of one or both of these latter conditions was found in seven of our patients. Saint's triad, the coexistence of hiatus hernia, cholelithiasis, and diverticulosis, was not discovered in any of our patients, although it has been recorded in 10% to 15% of patients in a large series.[11]

Indigestion, heartburn, postprandial epigastric or substernal distress, flatulence and nausea were present to some degree in all of our patients. A characteristic feature of hiatal hernia is the exacerbation of symptoms when lying down, particularly at night, or on bending forward. Either or both of these symptoms, found in all patients in this study, denote the presence of reflux œsophagitis and implicate a sliding component of the hiatus hernia.

Hiatal hernias tend to progress and develop symptoms and complications, particularly in the older age groups.[12] Reflux

œsophagitis is the commonest complication and symptomatically was present in all of our patients. Hæmorrhage, either occult or gross, occurred in nine cases. Frank ulceration was found in two patients. Incarceration of the hernia was present in three patients. Strangulation, gangrene and perforation is a rare but highly lethal complication[13] which did not occur in any patients in this series.

Treatment

The incidental finding of an asymptomatic hiatus hernia does not justify operative intervention. Size of the hernia and symptoms are unrelated. Conservative management includes postural instruction to prevent reflux œsophagitis, weight reduction when obesity is present, and an ulcer type of regimen. We have found the use of alkalis, particularly those which may be kept in the mouth and allowed to dissolve and trickle down the œsophagus, to be effective in the control of symptoms. Anticholinergics, such as Probanthine, are useful at bedtime to reduce gastric secretion at night.

Indications for surgical repair include increasing symptoms, lack of response to medical management, and the development of complications.[14]

Technique

Either a transabdominal or a transthoracic approach may be utilized in the repair of an œsophageal hiatus hernia.[1, 15, 16] Our preference is a transabdominal approach for these reasons:

1. We believe that reduction and repair of the hernia in most instances is easier, safer and more effectively performed through the abdomen. This is indirectly acknowledged by proponents of the thoracic route who frequently employ a counter incision in the diaphragm to allow access to the peritoneal cavity.[1, 7]

2. Exploration of the abdominal contents to exclude or treat concomitant disease is advisable, particularly when bleeding is a complication.

The transabdominal approach was utilized in all patients in this series except one. In this patient, the transthoracic approach was employed to determine the feasibility of using an Ivalon patch in the repair by this route. This technique was applicable by the transthoracic approach.

Satisfactory exposure was obtained in our patients with a high midline or left rectus incision. The left lateral ligament of the liver was divided, and the left hepatic lobe retracted medially.

The principles of surgical repair of an œsophageal hiatus hernia are reduction of the hernia, elimination of the sac and restoration of the anatomic functional integrity of the œsophageal hiatus. The hernia in our patients was readily reduced and the stomach replaced below the diaphragm. When a true hernial sac was found, the redundant peritoneum was either excised or utilized in the repair.

Restoration of an intact œsophageal hiatus required narrowing of the dilated hiatus, restoration of the oblique œsophagocardiac angle, and support of the repair to prevent recurrence. At operation the defect was most often in an oblique, and occasionally in a transverse plane (Fig. 2). The enlarged hiatal opening was reduced in size by approximation of the crura on both sides of the œsophagus. The accurate placement of these crural sutures restores

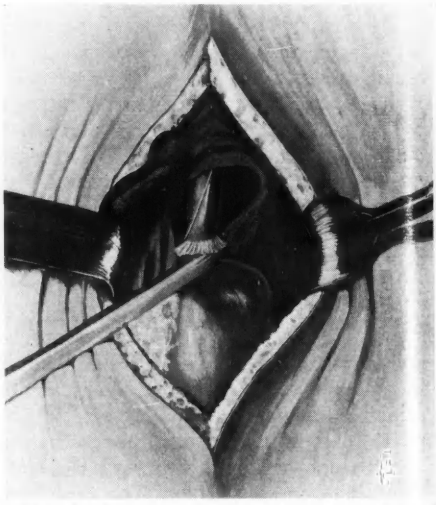


Fig. 2.—Drawing shows enlarged œsophageal hiatus. Œsophagocardiac junction is retracted inferiorly. Freed phrenœsophageal ligament presents an apron-like appearance.

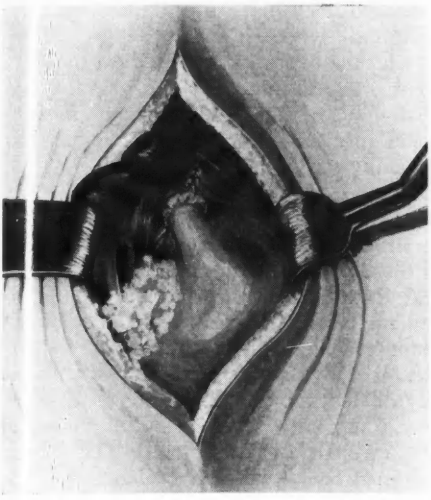


Fig. 3.—Stage of repair before insertion of prosthesis.

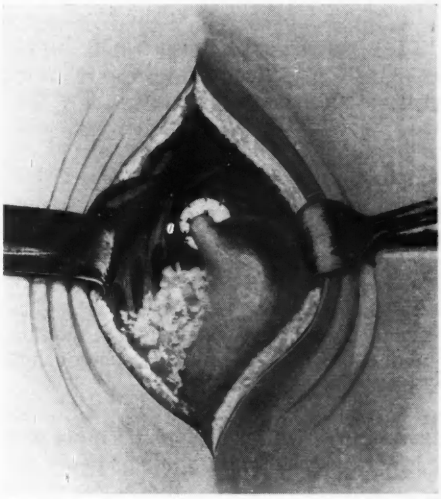


Fig. 5.—Completed repair showing Ivalon prosthesis sutured in position.

the oblique angle of the œsophagocardiac junction.

We believe that this muscular repair is insufficient in most instances. A fascial support was provided by suture of the cut edge of the phrenœsophageal ligament with or without peritoneum to the undersurface of the diaphragm (Fig. 3). This provided a restraining cuff around the distal œsophagus. Unfortunately, in many patients the phrenœsophageal ligament may be difficult or impossible to delineate, or it may be attenuated, friable and torn during the operative manipulation. In these patients, the use of a carefully placed prosthetic material as a support to the repair is of particular advantage.



Fig. 4.—Photograph of compressed Ivalon sponge cut into "C" pattern.

Polyvinyl formalinized sponge (Ivalon) was used experimentally in dogs for this purpose by Cooley, Grindlay and Clagett in 1957.[4] Their results were satisfactory.

The Ivalon used in our series is obtained as a porous block which is soaked and dried to eliminate any residual formalin. A portion of the sponge is then cut to the desired size, moistened, compressed in a metal press and autoclaved. These flat prostheses were cut in a C-shape at the time of operation to fit around the repaired hiatus on the under surface of the diaphragm (Fig. 4). With the open end of the "C" facing medially, the prosthesis was sutured in position. An outer layer of sutures provided anchorage to the diaphragm, and an inner layer of sutures attached it to the phrenœsophageal ligament or to the inner margin of the hiatus (Fig. 5).

This technique was utilized in 17 of those patients with œsophageal hiatus hernia who underwent surgical repair at the University of Alberta Hospital since 1955. External drainage of the operative area was employed initially, but was found to be unnecessary and was discontinued.

Results

There was no operative mortality in this series, and all patients have been followed up since the time of operation. Ten patients have remained completely well with no symptoms and three patients have symptoms attributable to other causes. The remaining four patients developed symptoms of dysphagia. Three of these patients early in the series required one bougie dilatation while in hospital, and were well thereafter. In only one patient was dysphagia a late complication at six months, and she also responded well to bougienage (Fig. 6).

Transient postoperative pneumonitis occurred in two patients. Ventral hernia was a late complication in three patients. In one of these, this was the third upper abdominal procedure and the second repair for hiatus hernia. In the other two obesity was a marked feature with failure to respond to a weight reduction regimen.

Discussion

The symptoms of œsophageal hiatus hernia in most patients are not due to the hernia *per se* but are caused by the œsophagitis associated with reflux of gastric contents. Ulceration, stenosis and hæmorrhage may develop. Surgical interventio: is indicated when a complication occ rs which fails to respond to medical mana ge-ment. Surgical morbidity and mortality re relatively low, and the results of mod rn techniques of direct hernial repair re generally satisfactory.

In 1957, Cooley, Grindlay and Clag tt[4] suggested the use of an Ivalon prosth sis to strengthen the standard surgical re air in those patients with marked enlargem nt and distortion of the œsophageal hia us, and associated weakness or absence of he phrenœsophageal ligament. In their exp ri-mental study in dogs the hiatus was ma u-ally dilated, the phrenœsophageal ligam nt was completely severed and a muscular crural repair was performed. This repair was reinforced with an Ivalon patch sutured to the under surface of the diaphragm and to the fascia propria of the œsophagus. In our patients the central portion of the Ivalon prosthesis was sutured to the phrenœsophageal ligament or the inner margin of the reconstructed hiatus, since direct attachment to the œsophagus was felt to be undesirable.

Ivalon has also been used successfully by Pesek and Keely[17] in 1958, to repair large diaphragmatic defects produced experimentally in dogs. There are numerous

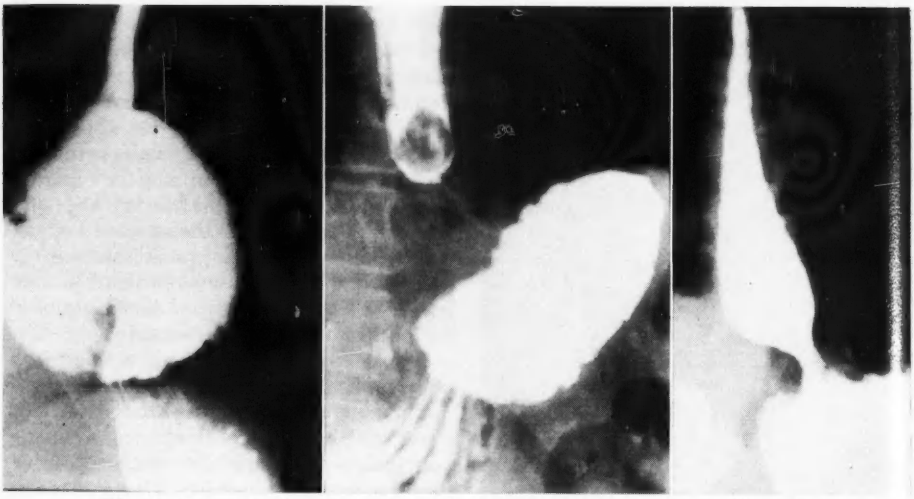


Fig. 6.—Composite barium meal roentgenogram; (left) preoperative film illustrating herniated stomach above diaphragm, (centre) postoperative film at six months shows narrowed distal œsophagus (note bolus of food in lower œsophagus), and (right) final film following bougie dilatation. Dysphagia has been relieved.

advantages to Ivalon sponge as a prosthetic material. It is relatively inert to tissue reaction, but acts as a scaffolding for the ingrowth of fibrous tissue with which it becomes incorporated. Ivalon is a tough resilient material which is pliable and readily sutured. It has little tendency to excite fluid collections or abscess formation.

The recurrence rate of hiatal hernias has varied a great deal in the literature, but has been reported as high as 40% by Husfeldt.[18] An encouraging feature of this preliminary report is the absence of recurrence at this time in our small series. However, the follow-up period is still too short to be of significance. Three patients are within one postoperative year, and 14 patients have been followed up from one to five years since operation.

It is obvious from this study that there are two complications which have occurred with greater frequency than one would expect or desire. Dysphagia was present early in the series when the prosthesis was applied too close to the œsophagus and the resulting hiatus was inadequate. This has been corrected in later repairs. By leaving a gap of about one-eighth of an inch between the œsophagus and the inner margin of the encircling prosthesis, stenosis and dysphagia can be avoided. The other complication, that of ventral hernia, may be eliminated by insisting on weight reduction before the operative procedure. An alternative method is the use of the transthoracic approach in very obese patients with recurrent hiatus hernia.[19]

Despite these complications, our results to date have generally been satisfactory. We believe that the clinical use of Ivalon sponge as an adjunct in the surgical repair of œsophageal hiatus hernia is worthy of consideration.

Summary

The use of a prosthetic material may be a valuable adjunct in the standard surgical repair of œsophageal hiatus hernia.

Compressed C-shaped patches of Ivalon sponge were utilized for this purpose in a series of 17 patients with hiatal hernia in whom operative repair was performed.

Results have been generally satisfactory to this time. Dysphagia developed as a transient postoperative complication in several patients, but can be avoided by careful placement of the prosthetic material.

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Résumé

La présente étude a pour but de réviser certains aspects anatomiques et physio-pathologiques des hernies de l'hiatus œsophagien et de présenter une série de 17 malades dont la correction chirurgicale a été additionnée d'une prosthèse d'Ivalon.

Postérieurement l'hiatus n'est relativement pas à l'abri où une pression aura tendance à l'élargir par séparation longitudinale des fibres du pilier droit. La projection péritonéale à la jonction cardio-œsophagienne qui était relativement lâche favorise la formation d'un sac herniaire.

Le rôle de l'hiatus est de permettre le passage du contenu œsophagien dans l'estomac et de prévenir le reflux gastrique. L'intégrité fonctionnelle est assurée par l'obliquité de la jonction œsophago-gastrique, par un mécanisme constricteur œsophagien probable, et par le support des piliers.

L'étiologie est indéterminée chez la plupart des malades porteurs d'hernie hiatale. Les syn)-tômes sont la plupart du temps dûs à l'œsophag e causée par le reflux gastrique. L'intervention c i-rurgicale est indiquée lorsque le traitement mé i-cal ne peut enrayer la complication.

Les principes de la réparation chirurgicale cc)-prennent la réduction de l'hernie, l'ablation tu sac et le rétablissement de l'intégrité anatomic te et fonctionnelle de l'hiatus œsophagien. L'orif e est rétréci par rapprochement des piliers diaph a-gmatiques, additionné de la juxtaposition du li a-ment phrénœsophagien à la face inférieure tu diaphragme. Dans bien des cas, ceci est de ré. i-sation difficile; d'où l'utilisation d'une proth se d'Ivalon en forme de "C" taillée et comprin e préalablement, qui est fixée par son bord conv e au diaphragme et par son bord concave, au li a-ment phrénœsophagien (Fig. 5).

Cette technique a été utilisée chez 17 mala es et les résultats ont été généralement bons. On observa de la dysphagie post-opératoire transito re chez quelques patients, laquelle peut être élimin e par la mise en place minutieuse de la prothèse.

URINARY DIVERSION*

"Broadly considered, the term urinary diversion indicates the changing of the normal course of urinary flow from the kidney to the body exterior by surgical alterations of its proper channels, or by conversion to others, surgically created for the purpose or both. Technical procedures for its accomplishment may be limited and simple or extensive and complex. The simple limited techniques (for example, urethrotomy, cystotomy, cutaneous ureterostomy and nephrostomy), are in the main familiar because of early development and long usage. The more recently devised diversionary procedures are not, because to a large extent they are still in the category of surgical innovations, and concerning them there is less general knowledge and considerable speculation. As their usage increases, some of them will no doubt be discarded and others accepted and standardized according to particular indications.

"It is the purpose of this writing to examine as comprehensively as possible, certain of the more promising of recently developed techniques for urinary diversion. This is done in the hope that such will prove of value to some in the management of disorders where the procedure may be essential to palliation for cure.

"GENERAL CONSIDERATIONS. It may be generally stated that while the procedures for urinary diversion have changed, the indications for its performance have not. These indications may be largely included under a single indication with a few additional ones. This is actual or impending obstruction of urinary flow through its normal channels. Congenital anomalies, inflammation, metabolic disorders, and neoplasms may all produce obstruction of the various parts of the urinary tract. These causes represent theoretically an almost limitless variety of etiological agents of urinary obstruction, all having the common feature of impending or occluding urinary flow through the various components of the urinary tract. There are, however, other indications for diversion, far less frequently encountered which are not obstructive in character. One of these is in conditions where diversion of urinary flow is advisable to create conditions optimal for healing of lesions incident to trauma or inflammation. The other is for the relief of disorders in which severe pain is produced by urinary flow through the normal channels. Thus, the principal indications for urinary diversion may be included under one, a consideration of any two, or all three of the following: (1) relief of impending or actual obstruction, (2) the promotion of healing, (3) the elimination of pain. . . ."

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*Crowley, R. T. and Swigart, L. L.: *Am. J. Med. Sc.*, **240** (No. 2): 232, 1960.

HYDROCEPHALUS: A SHUNT BETWEEN THE VENTRICLE AND STENSON'S DUCT

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A EXCELLENT review of the various shunting procedures employed to date in the surgical treatment of hydrocephalus, with their advantages and disadvantages, has recently been published.[1]

Amelioration of the hydrocephalic head is difficult to assess. There are no standard measures of anticipated progress for any given type of case; hence, short term or long term improvements are related with difficulty to therapeutic efforts. Occasionally, lesions arrest spontaneously or with no other treatment than repeated lumbar punctures, or maintain an arrest after removal of a shunt no longer functioning. Hence we are at present without data regarding the end-result of various types and degrees of hydrocephalus with or without shunts for any given length of time.

The concept of shunting the hydrocephalic fluid into Stenson's duct developed during a casual conversation with Dr. Harold Rice some eight years ago. The advantages are that this is a surgically simple procedure without sacrifice of a vital organ and without loss of body fluid or electrolytes.

In the approach to this proposed procedure several unanswered questions presented. It was unknown at what age Stenson's duct became patent and at what age the parotid glands began to function. It was unknown what would happen to the normal parotid gland if the duct were ligated. It was not known how long a duct would tolerate a polythene tube nor whether it would tolerate the cerebrospinal fluid bathing its mucosa. It was not known at what head of pressure the muscular opening of Stenson's duct would relax. A search of the literature and interrogation of surgical, anatomical, and physiological colleagues failed to provide answers to these problems. Several experiments were performed on dogs, and numerous stillborn infants were dissected over a period of three years. It was found that at the age of three months the human parotid duct was microscopically patent and at seven months the duct had a lumen which would admit a No. 22 needle. The greatest concern was the unknown behaviour of the parotid gland following ligature of the duct. The possibility of parotitis with infection ascending up the tube to the brain, held our hand in attempting this procedure on humans, although we demonstrated that in dogs, ligature resulted in no sequelae other than a very prompt atrophy of the gland.

Surgical Procedure

A small incision is made above the zygoma just in front of the ear to accommodate burr hole entrance into the dilated temporal horn. A larger curved incision is then made below the zygoma in front of the ear, exposing the pyramidal portion of the parotid gland, the facial nerve branches and the parotid duct. Initially one might have difficulty in finding Stenson's duct in an infant. It was found at a much lower level than anticipated from the anatomy books and was only located after rolling out the buccal fat pad from in front of the masseter muscle. The duct was then clearly visible coursing back over the surface of the masseter into the pyramidal projection of the parotid gland. It lies deep to the seventh nerve branches but these posed no problem in any of the exposures to date. As the duct is incised it can be seen to constrict to an extent that the lumen becomes invisible. After waiting a few moments the lumen relaxes and a polythene tube which will accommodate a No. 22 needle can be teased into the lumen using a technique similar to that of threading an indwelling catheter in a baby's cutdown. It has been our practice to create a small bend in this tube. This is done by threading a wire stent into the tube, bending both to the desired shape, then placing both tube and wire in boiling water for a minute and then in cold water. The wire is then

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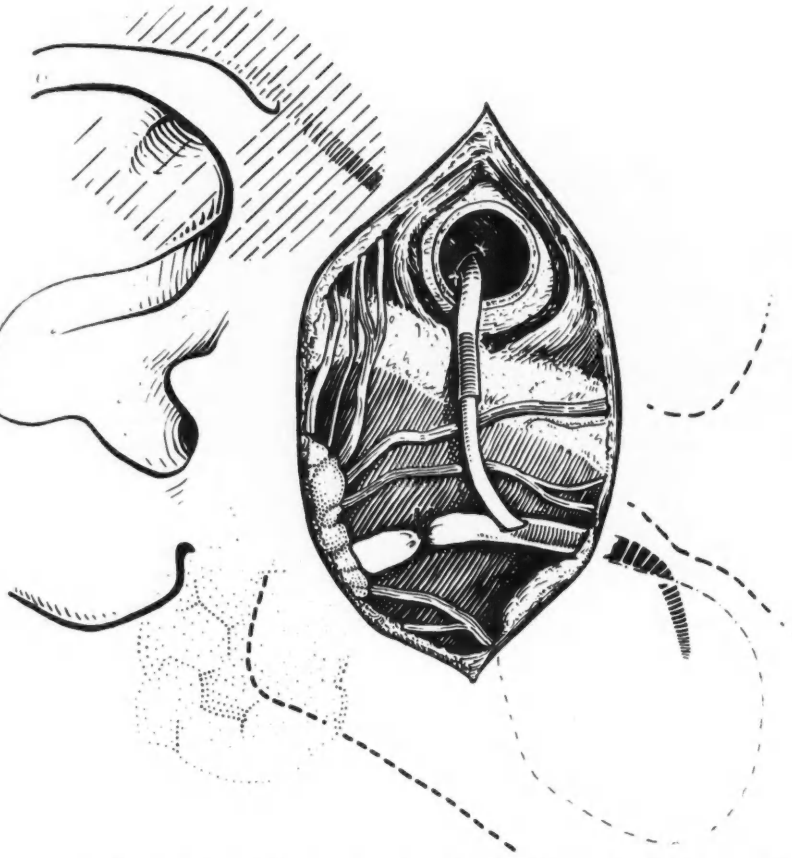


Fig. 1.—Drawing of surgical exposure with ear to left and burr hole just above zygoma. Note the telescoped polythene tube passing over the facial nerve branches before entering the ligated parotid duct just before it passes beneath the buccal fat pad indicated by the outline with lines and dots.

removed and the tube resumes and retains the desired curvature. The tube is firmly anchored with silk sutures after which a larger tube which will accommodate a No. 18 needle, is anchored into the ventricle by suturing it to the dura and threading it through a tunnel between the two incisions. The patency of both ends of this shunt can now be tested *in situ*, without undue manipulation. The tubes are then cut to the appropriate length and fitted snugly together without unnecessary manipulation of either anchored end. The surgeon is thus assured of the patency of the system with a minimal amount of final manipulation (Fig. 1).

It was noted at operation that a column of water approximately 80 mm. in height would build up before Stenson's duct relaxed, after which there would be a very free rundown to a level of approximately 30 mm. This of course, was in the anæsthetized patient.

Three human patients have been subjected to this procedure to date; two infants who were virtually hydrancephalic with complete transillumination of the head, and one adult in terminal condition with a midbrain tumour. In all three the shunt functioned perfectly for approximately three months, and then ceased to function. At autopsy the lumen of Stenson's

duct was occluded with granulation tissue just distal to the end of the polythene tube. It is not known whether this granulation tissue developed in response to the presence of the polythene tube or to the presence of the spinal fluid bathing the duct surface.

In summary, a ventriculo-Stenson's shunt has the advantage of surgical simplicity without sacrifice of any vital organ and with preservation of electrolytes. As yet it has not been demonstrated that Stenson's duct will tolerate a tube and/or cerebrospinal fluid for any significant length of time. It is planned to investigate the suitability of other materials as they become available at the required calibre, and it is hoped that other surgeons may consider investigation of this procedure and improve its technique.

Summary

Experience with shunting of spinal fluid from hydrocephalic ventricles to Stenson's duct is presented. This route has the advantage of surgical simplicity, without sacrifice of a vital organ or loss of body electrolytes. However, to date none of these shunts has functioned for more than three months. Their failure has been due to occlusion of the duct by granulation tissue distal to the tip of the polythene shunt. It is not known whether this reaction is due to the presence of the spinal fluid or to the polythene foreign body.

Acknowledgment

The assistance of the medical artist, Miss Nancy Joy, is gratefully acknowledged.

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Résumé

L'évaluation des traitements palliatifs de l'hydrocéphalie présente quelques difficultés puisque l'histoire naturelle de cette affection n'est pas encore complètement connue. Dans certains cas on observe un arrêt spontané dans l'évolution de la lésion à la suite de ponctions lombaires répétées ou même longtemps après le blocage d'un drain de dérivation. L'intervention décrite ici consiste à établir une communication entre une corne temporale et la bouche par le truchement d'un tube de polythène relié au canal de Stenson. La voie d'accès emprunte une petite incision au-dessus du zygoma immédiatement en avant de l'oreille. Il s'agit en réalité de deux tubes de calibres légèrement différents; le plus petit est inséré dans le canal et fixé en place, l'autre, relié au ventricule par un petit volet, est solidement attaché à la dure-mère. Après s'être assuré de la perméabilité de la lumière de chacun, l'opérateur réunit les deux bouts libres en les insérant l'un dans l'autre. Dans trois cas où il en a fait l'application, l'auteur a constaté que cet appareil a fonctionné pendant environ trois mois après quoi la lumière du canal devint oblitérée par du tissu de granulation. Il n'a pu établir si cette réaction de l'épithélium du Stenson venait du contact avec le polythène ou de la présence de liquide céphalorachidien. Le procédé se recommande par sa simplicité.

PHONOCARDIOGRAPHIC FEATURES OF ATRIAL SEPTAL DEFECT*

". . . The development of safe and effective surgical technics for the closure of atrial septal defects has increased the importance of accurate preoperative diagnosis. This lesion is common and, especially in younger patients, the physical signs may occasionally simulate those of a normal patient with a functional systolic murmur. Ancillary diagnostic methods that might obviate the need for cardiac catheterization would therefore be valuable. Atrial septal defect is characterized by a large left-to-right shunt with increased blood flow across the tricuspid and pulmonic valves and by delayed electrical depolarization of the right ventricle resulting usually in a pattern of incomplete right bundle-branch block in the electrocardiogram. Since both these features may produce distinctive changes in the heart sounds, phonocardiography may be an important technic in the correct diagnosis of this lesion. . . ."

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ENTEROGENOUS CYSTS OF THE DUODENUM*

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ENTEROGENOUS cysts of the duodenum represent a rare developmental abnormality. Gross[10] and other authors included them correctly in the entity of duplications of the alimentary tract. However, enterogenous cysts of the duodenum deserve special consideration, due to two factors: (1) Their anatomical location and differences in surgical treatment related thereto, and (2) their relationship to the biliary system and possible significance of this in the physiopathology of biliary drainage into the duodenum.

EMBRYOLOGICAL CONSIDERATIONS

In order to be designated as a true enterogenous cyst, this developmental abnormality must exhibit, according to Gross[10] and Du Toit,[7] the following characteristics: (a) It should be attached to the segment of the alimentary tract in question; (b) it must possess a smooth muscle coat; (c) it should be lined by mucosal epithelium resembling that of the alimentary tract, although not necessarily that portion which is adjacent to it. The majority of enterogenous cysts of the duodenum reported so far were intramural and it appears that the suggestion of Lewis and Thyng[12] and Bremer[5] as to the origin of intestinal duplications is most applicable in this respect.

Lewis and Thyng[12] based their theory on the minute epithelial buds, located chiefly on the antimesenteric aspect of the primitive gut, which can be observed in embryos at the end of the second month; these can unite with the intestinal lumen by a stalk or become completely detached. Normally these epithelial elements become completely obliterated. Continuous evolution and secretory function of these rests lead to cystic formation.

Bremer's theory[5] is based on observations in six to seven-week-old embryos, in which he observed active proliferation of the intestinal lumen, i.e. the so-called "solid stage". The "solid stage" never involves the entire primitive gut but is distributed rather segmentally.[3] The epithelial cells secrete droplets of fluid which form intercellular vacuoles. Coalescence of these vacuoles re-establishes the lumen of the gut. It appears feasible that some of these epithelial elements may fail to merge with the intestinal lumen and subsequently form an enterogenous cyst. Gross[10] emphasized the fact that duplication of the alimentary tract may occur between various layers of the wall or may be immediately adjacent to the bowel wall. In the case of enterogenous cysts of the duodenum, the usual location of the cystic structure is intramural.

CLINICAL AND PATHOLOGICAL DATA

Enterogenous cysts of the duodenum are found much less frequently than benign tumours of the duodenum, a group of lesions of immediate importance as far as differential diagnosis is concerned. Alsobrook[2] compiled a survey of 80 cases of benign duodenal tumours (including adenomas, myomas, pancreatic cysts, and rare tumours arising from various elements of the duodenal wall) and observed one case of an enterogenous cyst of the duodenum. In the series of duplications of the alimentary tract reported by Gross[10] the incidence of enterogenous cysts was approximately 2%. The most frequent site reported in the literature appears to be the second portion of the duodenum, although cases of cysts of the first portion have also been reported.[14] Usually one cyst is present, although Peple[14] found two enterogenous cysts located adjacently. The enterogenous cysts are commonly oval in shape, their size varying from 4 cm. to 8 cm. in the longitudinal diameter. Their usual position is at the mesial aspect of the gut, although they have been found also in the lateral wall. The wall of the cyst consists of a layer of epithelium with various amount

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of underlying smooth muscle surrounded by a thin fibrous layer. In the portion of the cyst adjacent to the lumen, this arrangement produces a double layer of intestinal epithelium, with interposed smooth muscle elements. The epithelial lining of the cyst is similar to that of the duodenum. The epithelium may be atrophic due to intra-luminal pressure effects in larger cysts or it may exhibit inflammatory and hemorrhagic changes. The content of the enterogenous cysts of the duodenum is usually a clear, mucoid secretion, although in several cases, bile[6] and even biliary calculi have been found.[9, 15]

The clinical symptoms are not typical. Most patients complain of pain in the epigastrium or right hypochondrium, occasionally radiating to the back. The pain is not definitely related to food intake, although it usually occurs at least one hour after meals or later. Nausea and vomiting are almost uniformly encountered in these patients, the degree of severity of symptoms depending on the extent of duodenal obstruction and interference with passage of food through the duodenum.

Generally, the symptoms are intermittent in character and sometimes can be traced to infancy. The vast majority of reported cases occurred in children and young individuals, although cases have been reported up to the 69th year of age.

Radiological Findings

Radiological examination following a barium meal discloses an ovoid filling defect of smooth and regular outline, usually located in the posteromedial aspect of the descending portion of the duodenum. The duodenal segment in question appears dilated, with patency of the intestinal passage preserved along the lateral border of the cyst. The enterogenous cyst usually exhibits some elasticity and slight compression of the cyst wall can be observed on passage of the food bolus. The duodenal mucosal pattern is well preserved and shows no evidence of the central umbilication typical of many cases of aberrant pancreatic tissue and connective tissue tumours.

The differentiation from various benign duodenal tumours is difficult and sometimes impossible. The chief radiological sign of importance appears to be a certain elasticity and compressibility of the cyst wall, which is related to the liquidity of contents. Differentiation from malignant lesions of this region usually presents no problem: in the latter the fixation, irregularity or destruction of the contour are typical.

Surgical Treatment

The most satisfactory treatment of enterogenous cysts of the duodenum is complete excision. The cyst can be approached by longitudinal duodenectomy of the anterolateral wall and incision of the mucosa of the duodenum overlying the cyst. The cyst is then separated by blunt dissection from the adjacent elements of the duodenal wall. Particular attention should be paid to any ductal or stringy elements entering the cyst wall, as communication with the biliary tree has been described, and communication with the pancreatic ductal system is possible.

It seems advisable to implant the opening of any undiagnosed ductal structures into the duodenal lumen. Following the incision, the intramural space is obliterated by approximation of the mucosal layer with interrupted catgut sutures.

Excision of the cyst is not always possible because of its size or proximity to the common bile duct or pancreatic duct openings. In such cases, the best policy appears to be that adopted by Gardner and Hart[8] who established a wide window in the duodenal wall overlying the cyst to ensure adequate and permanent drainage of the cyst contents into the intestinal lumen. Gross[10] has emphasized that the "window drainage" should be not only of ample width, but also located preferably in the most dependent portion of the cyst.

Following this procedure, the Wangensteen suction tube can be usually manipulated into the duodenal lumen to ensure decompression of the adjacent segment of the gut. The anterior duodenal wall incision is then closed transversely by two layers of sutures. In cases with extensive cysts and difficult dissection, establishment



Fig. 1.—Roentgenogram taken in erect position before operation. A large oval-shaped filling defect in the second portion of the duodenum is visible. Deformity of the duodenal cap, related to a peptic ulcer, is also present.

of a gastroenterostomy may be indicated.

Co-existence of the cyst with a gastric or duodenal ulcer merits particular consideration. Such an association was found in one of the reported cases, as well as in



Fig. 2.—Roentgenogram taken in prone position before operation. Note the regular outline of the enterogenous cyst.



Fig. 3.—Posterior-anterior projection, with patient in prone position. The mucosal pattern of the duodenum is preserved; it is normal in the upper segment of the second duodenal portion and stretched by the cyst in the lower segment.

the case presented in this publication. In both instances the cyst was located in the duodenum, interposed between the papilla of Vater and the ulcer. It is therefore possible that some interference with biliary reflux into the duodenum acted as a contributing factor in the ulcer formation.

Review of Recently Recorded Cases

Mendl and Tanner[13] recorded 20 cases up to 1953. Polson and Isaac[15] were able to locate two further cases and added one of their own. We were able to locate four additional cases published subsequently in the literature,[1, 4, 6, 11] so that a total number of 27 have been reported on to date. The most recently reported cases presented the following features. A two-month-old baby with a large enterogenous cyst involving the first and second duodenal portion was reported by Adams.[1] Barooty *et al.*[4] described a 19-year-old girl with a solitary cyst of the descending portion of the duodenum. In DuBerger's case[6] the cyst of the second duodenal portion communicated with the biliary system and con-

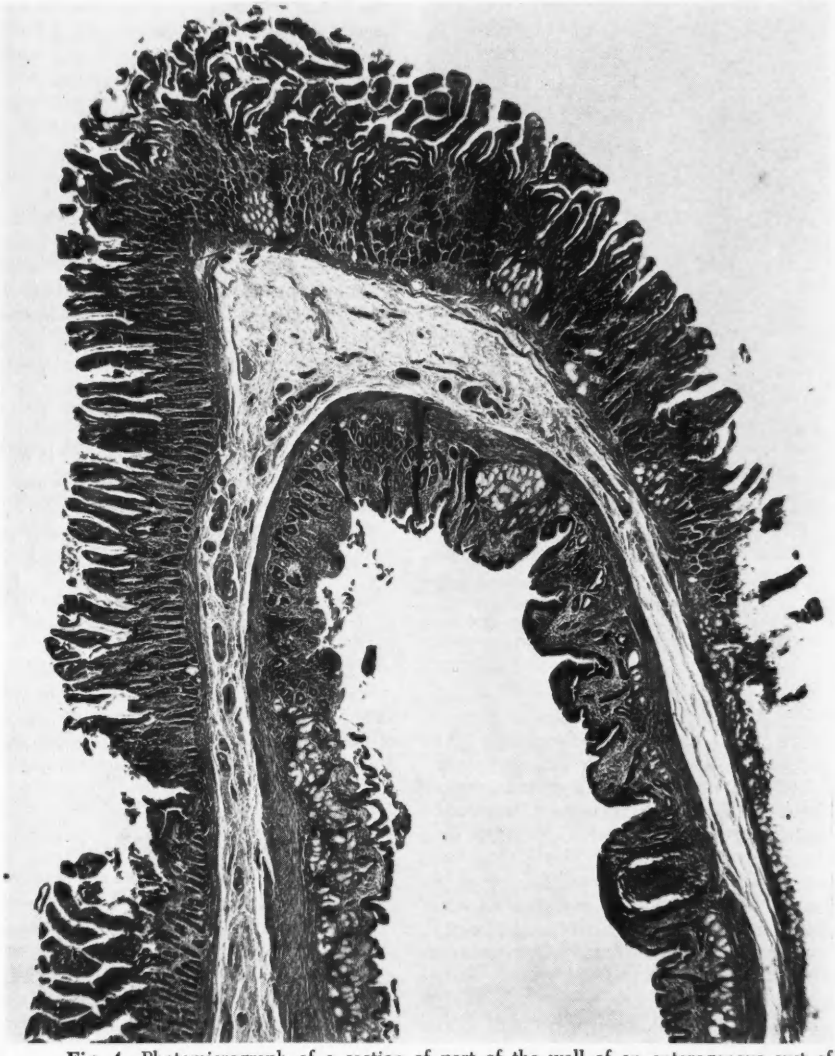


Fig. 4.—Photomicrograph of a section of part of the wall of an enterogenous cyst of the duodenum. The normal duodenal wall is on the periphery and includes mucosa, muscularis mucosae, submucosa and muscle wall. The central cavity represents the enterogenous cyst, which is lined by a duodenal type mucosa resting directly on the muscle coat of the duodenal wall.

tained bile. Other cases of communication of the enterogenous cyst with the biliary duct were those of Gordimer and Bluestone[9] and Polson and Isaac;[15] in both, biliary calculi were present in the cystic lumen. Kirtley and Matuska[11] described the case of a 40-year-old man with an enterogenous cyst of the descending duodenum.

Case Report

The history of a case of enterogenous cyst encountered recently in St. Mary's Memorial Hospital follows: a 25-year-old white man was admitted to the hospital with a history of intermittent upper abdominal distress of two years' duration. The pain did not appear to be definitely related to food intake, though it had never occurred earlier than an hour



Fig. 5.—Roentgenogram in prone position, taken eight weeks after operation. The second duodenal portion is of normal appearance. The duodenal cap is seen in a phase of contraction; no ulcer crater is present.

following a meal. It was localized in the epigastrium and right hypochondrium. The patient stated that he frequently felt nauseated but did not vomit. Six months before admission to hospital, the patient had radiographs taken elsewhere and a diagnosis of a duodenal ulcer was made. The patient was placed on a standard ulcer regimen and medication, without subsequent evidence of ulcer healing. On admission to St. Mary's Hospital, physical examination disclosed right epigastric tenderness on palpation. Total gastric acidity was increased, but free acid was within normal limits. Radiological investigation demonstrated an oval-shaped filling defect on the medial aspect of the descending portion of the duodenum, which could be observed following a barium meal. The defect was characterized by a smooth outline and relative compressibility on passage of the contrast medium. The patency of the duodenum was preserved along the anterior and lateral borders, though the lumen was partially obstructed; the duodenum was somewhat dilated at this level and its lumen was narrowed by the mass. The radiological diagnosis was summarized as follows: A benign intramural, space-occupying lesion of the descending portion of the duodenum, enterogenous cyst being the first diagnostic possibility. In addition, a small duodenal ulcer with deformity of the duodenal cap was found.

A right paramedial laparotomy was performed shortly afterwards. A cystic mass could be easily palpated in the duodenum before it was opened by a longitudinal incision in the anterior wall of the descending portion. On palpation the cyst appeared movable within the wall; after incision of the duodenal mucosa of the posteromedial wall, the cyst, measuring approximately 5 cm. x 2 cm. was dissected bluntly from the surrounding tissues. A narrow stalk on the lateral aspect of the cyst was encountered; though no lumen could be demonstrated by probing, this was implanted into the duodenal lumen following removal of the cyst and closure of the defect with interrupted sutures. A Wangensteen suction tube was then passed through the pylorus. The anterior duodenal wall was closed transversely with two layers of sutures.

The postoperative course was uneventful and the patient left hospital 10 days later. He has had a barium meal two and six months postoperatively, which disclosed residual scarring of a healed duodenal ulcer and a perfectly patent duodenum.

The pathological report was that of an enterogenous cyst of the duodenum with a characteristic back-to-back positioning of the mucosal layers of the cyst and the duodenum. It could not be decided with certainty whether the symptoms presented by this patient were related to the duodenal ulcer or to the enterogenous cyst. The duration of symptoms (two years) suggests that these were due to the ulceration; the cyst had probably been present for a long period of time and was asymptomatic. The patient was operated upon because of the presence of an obstructive lesion of the second portion of the duodenum. The duodenal ulcer did not respond to a six-month period of intensive medical and dietary management and healed only after removal of the enterogenous cyst. This suggests that the cyst caused an obstacle to the biliary reflux into the duodenal cap and in this way probably interfered with the healing of the ulcer.

It appears that enterogenous cysts of the duodenum arise from the malformation and continuous development of epithelial intestinal elements in the duodenal wall. The occasionally observed communication with the biliary passages, as found in cases reported by Gordimer and Bluestone,[9] Polson and Isaac[15] and DuBerger[6] suggest

that abnormalities of vacuolation of the primitive gut may persist until the biliary passages develop, with subsequent establishment of an abnormal communication. Similar relation to the pancreatic drainage system or aberrant pancreatic tissue might be postulated, although it has never been reported. The suggestion that such cysts may arise directly from aberrant pancreatic tissue appears unlikely in view of the invariably reported intestinal mucosal lining of the duodenal cyst wall.

The diagnosis of the condition rests primarily with the radiologist, in view of the rather indefinite and atypical symptomatology. Familiarity with the character of the lesion might enable a cure by a relatively simple surgical procedure.

Summary

The subject of enterogenous cysts of the duodenum is reviewed on the basis of 27 previously reported cases and one case operated on in St. Mary's Memorial Hospital, Montreal. The embryogenetic origin of the enterogenous cyst of the duodenum is discussed. It is pointed out that the diagnosis of an enterogenous cyst of the duodenum can be suggested radiologically following a barium meal. The surgical treatment consists of transduodenal excision of the cyst with obliteration of the intramural space or establishment of "window drainage" when excision is not feasible. The possible interference of the cyst with biliary reflux and ulcer healing is suggested.

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Résumé

Une révision du problème des kystes entérogènes du duodénum: étude de 27 cas déjà publiés et d'un autre au "St. Mary's Memorial Hospital" de Montréal.

L'origine embryonnaire est discutée. On fait remarquer que son diagnostic peut être suggéré radiologiquement par repas baryté. Le traitement chirurgical consiste en son excision trans-duodénale avec oblitération de l'espace intramural, ou l'élargissement de la lumière duodénale (si l'excision est impossible). L'interférence possible du kyste avec le reflux biliaire et la guérison d'un ulcère est mentionnée.

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THE NAIL-PATELLA SYNDROME: (HEREDITARY ONYCHO-MESODYSPLASIA)

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THIS CLINICAL entity has been largely ignored in orthopædic literature. It has not been recorded previously in Canada. The only recent publication on this continent was that by Love and Beiler[15] in 1957, who reported their study of a family in which 15 members in five generations, showed varying forms of the syndrome. The interesting and widespread nature of the lesions is considered worthy of an additional report.

REVIEW OF THE LITERATURE

In 1897, Little[14] collected from the literature 42 cases of congenital absence or delayed development of the patella, and added three further cases of his own. In his article he mentioned a family observed by William Sedgwick,[23] in which, in four generations, 18 members of the family had no patella and no thumb nail. It is not clear, however, if these two abnormalities were associated, or whether they occurred independently of each other. This is the earliest reference the author could find to an association between defects of the patella and of the nails.

In 1900, Wolf[32] reported a family in which the mother showed congenital absence of the patella and complete absence of both thumb nails. These two deformities were inherited by her children, but her grandchildren were normal.

In 1903, Most[17] described a family in which the absence of the thumb nails and extensive atrophy of the remaining nails was inherited by all six sons. One of these children is described by Senturia and Senturia[24] in 1944. This patient had five sisters, of whom four were carriers of the nail anomaly.

In 1912, Firth[4] reported an additional family and commented that the two deformities were either both present or both were absent. In addition, he noted that the mother and two children showed inability to fully supinate the forearm or extend the elbow joint. In his article he also recorded a family observed by Thomson[26] of Edinburgh, in which the mother was unable to completely extend the elbow joint. This is the earliest reference the author could find to the triad of abnormal nails, patellae and elbow joints.

In 1915, Rubin[22] reported a further family with this syndrome of developmental abnormalities.

Between 1915 and 1938, several references were made to hereditary nail dystrophy, with or without other abnormalities. Barrett[2] in 1919, recorded an association between nail defects and hereditary hypothyroidism. He also referred to other associated ectodermal defects such as icthyosis, keratosis of the palms, hypertrichosis, and premature or late eruption of the teeth. Tobias[27] in 1925, reported a family showing hereditary dystrophy of the



Fig. 1a.—Roentgenogram reveals increased thickness of the vault of the skull and some calcification of the falx cerebri.



Fig. 1b.—The lateral roentgenogram of the skull confirms the increased thickness of the vault and hyperostosis frontalis interna.



Fig. 1c.—The lateral roentgenogram of the cervical spine suggests that the arch of the atlas is bifid and is slightly above Chamberlain's line. Some degree of basilar invagination is probably present.

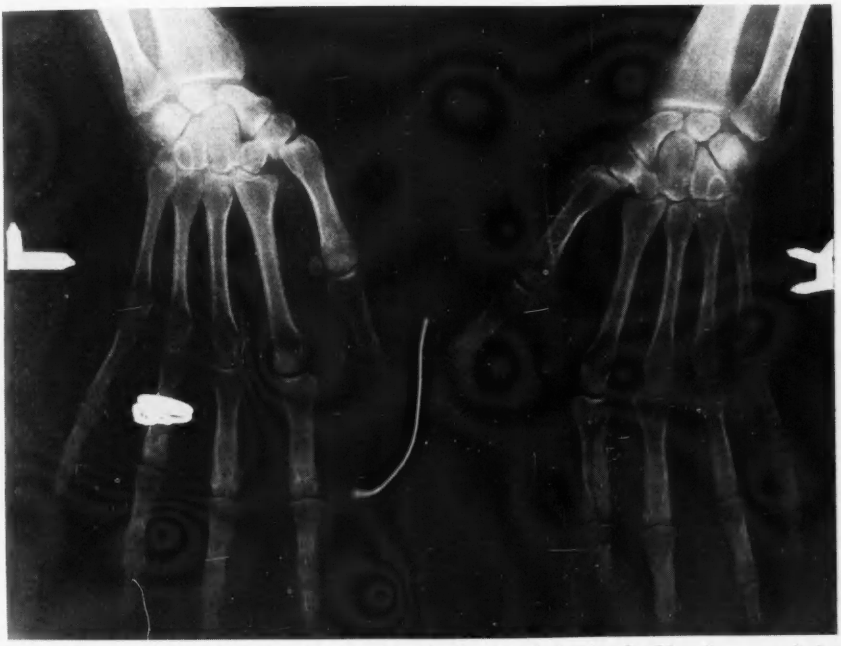


Fig. 2a.—Roentgenogram reveals that the bones of the hands (like the rest of the skeleton, with the exception of the skull) are poorly mineralized. The index metacarpal is the longest of the metacarpals. There appears to be a coalition between the trapezium and the trapezoid. The radial and ulnar styloids are at the same level.

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nails unassociated with any other defects. In 1928, Jacobsen,[7] in reviewing the literature, found four families in which the nail dystrophy was associated with congenital alopecia. Genetic studies of these conditions by Lees *et al.*[12] in 1957, have shown that nail and digital abnormalities are produced by an autosomal gene, separate and distinct from that involved in the syndrome under review.

In 1925, Trauner and Rieger[28] emphasized the dominance of luxation of the radial head in their report of the condition, inherited by six members of a family in four generations. They pointed out in addition, the associated hypoplasia of the capitellum of the humerus, and also recorded flexor contractures of the fingers.

Further reports by Oesterreicher[18] in 1930, and Aschner[1] in 1934, emphasized the three associated defects of the nails, patella and elbow joints.

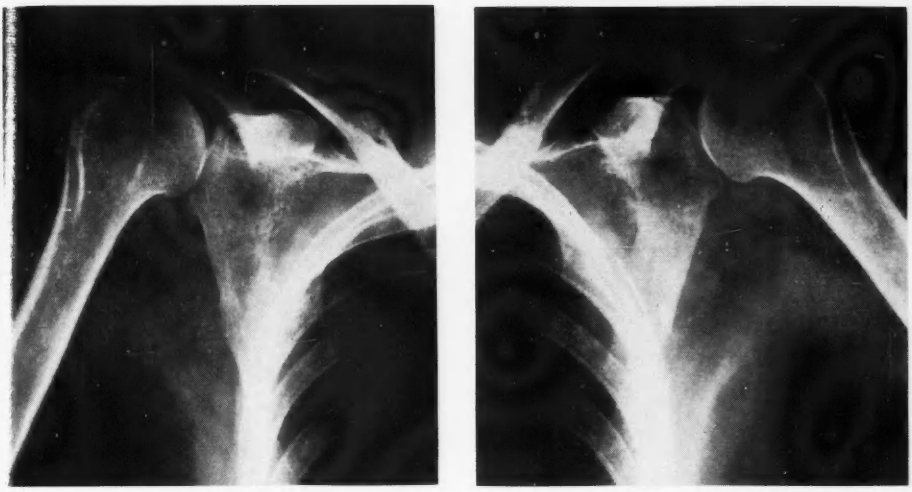
Turner[29] in 1933 gave a very clear description of the full syndrome. He added many other abnormalities which may coexist with those previously described. He pointed out the abnormalities which may be present in the region of the shoulder joint, such as prominence of the acromion and hypoplasia of the coracoid process and the anterior position of the humeral head. He also described in addition to the patella



Fig. 2b.—Roentgenograms reveal normal bones of the thumb, in spite of the lack of thumb nails, which is seen in Fig. 2c.



Fig. 2c.—Photographs show the abnormalities of the fingers. The thumb nails are completely absent and the nails of the indices are markedly convex. The distal skin creases on the fingers are poorly defined.



Figs. 3a and b.—Roentgenograms show both scapular regions. On each side the appearances are similar. There is hypoplasia of the neck, glenoid cavity and of the coracoid process. The outer body of the scapula is increased in convexity. The acromion is long and pointed.

defects, other changes which may occur in the region of the knee, such as undue prominence of the medial femoral condyle, increased medial bowing of the upper end of the tibia, and prominence of the tibial tubercle. He described additional features including a hump on the posterior surface of the lower end of the humerus and excessive extension at the metacarpophalangeal and interphalangeal joints. In conclusion, he suggested that the entire skeletal system was probably affected in this condition.

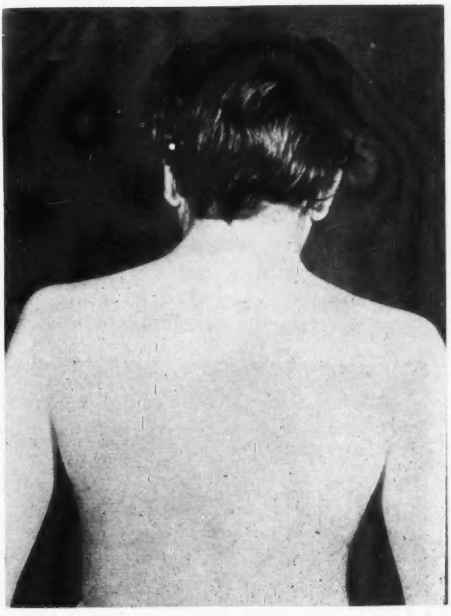


Fig. 3c.—Photograph shows the shortness of the neck which is accentuated by the low hair line. The shoulders are wide and square.

Lester[13] in 1936, recorded a family, first observed by Firth, which showed some additional abnormalities. The most unusual of these was a defect of the iris, which was cloverleaf in shape, with a clear-cut paler zone around its margin.

Montant and Eggerman[16] in 1937, and Sever[25] in 1938, noted a relationship between this syndrome and blue eyes and blonde hair.

In recent years, Jameson[8] and Lawler[10, 11] and their co-workers have carried out extensive genetic studies of families exhibiting features of the syndrome. They have accentuated the symmetrical nature of the lesions, particularly those involving the nails. They refer to the work of Hawkins and Smith[6] who noted that the syndrome predisposed to renal changes. These workers found a high incidence of orthostatic proteinuria in affected individuals. In one case coming to autopsy, a typical

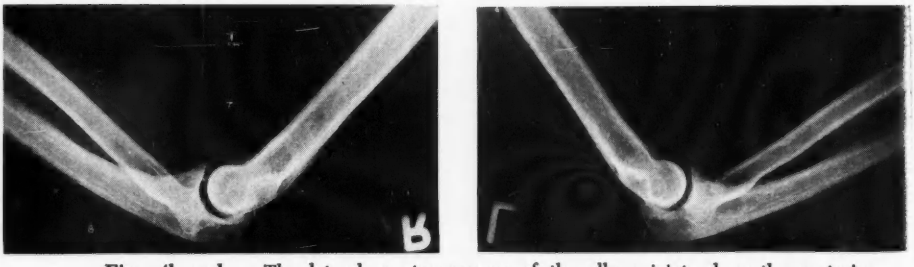


Fig. 4a.—Roentgenograms show both elbow joints. There is hypoplasia of the capitellum, hypoplasia, irregularity and posterior luxation of the radial head. The internal epicondyle is prominent and the carrying angle is increased.

glomerulonephritis was found, though the upper poles of both kidneys were apparently normal. Brixey and Burke[3] in 1950, and Rilliet[21] in 1953, also recorded the kidney defects.

Pelvic deformities are mentioned in many cases. Turner[29] in 1933, mentioned increased concavity of the external aspect of the ilium and prolongation of the anterior superior iliac spine. In his article he has reproduced a radiograph which, although not very clear, does show the typical "iliac horns" which were later described by Keiser[9] in 1939, and by Fong[5] in 1946. These pelvic deformities are referred to by Brixey and Burke[3] in 1950, and by Wedler and Welsch[30] in 1952. Jameson *et al.*[8] mentioned in their publication that the "iliac horns" are present in 30% of affected families.

Wildervanck[31] in 1950, described the syndrome occurring in five generations. The family consisted of 53 members, 22 of whom showed the different abnormalities



Figs. 4b and c.—The lateral roentgenograms of the elbow joints show the posterior luxation and hypoplasia of the radial head. Bony spikes are present around the coronoid process.



Fig. 4d.—Photograph shows the forearms and hands. The forearms are seen to be held in the mid-prone position. The ulna can be seen to be bowed posteriorly in its lower third.

in various combinations. He also recorded the presence of exostosis around the upper end of the ulna.

Case Report

Mrs. L.F., 52 years of age was of quite pleasant appearance, five feet two inches in height, and weighed 116 lb.

Family history.—It is reported by the patient under review that both her mother and grandmother, and many other close relatives exhibited similar abnormalities. Her only daughter, who has also been seen, and one of the daughter's sons, is also affected.

Past history.—Apart from a hysterectomy for menorrhagia 12 years ago, and recent symptoms of angina pectoris, she has been in good health, and for the past seven years has worked as a waitress.

Present complaints.—The patient was admitted to hospital for investigation of angina pectoris. She made no voluntary comment about her other disabilities, but on questioning she admitted to weakness of her knees and clumsiness of her thumbs and fingers. These disabilities had not prevented her from working full time as a waitress.

Physical examination.—No significant abnormality was noted on examination of the central nervous, the respiratory, alimentary or urinary systems. A recent electrocardiogram was considered to be within normal limits. The blood pressure was 136/68 mm. Hg.

The skin.—This was of normal elasticity. A single lower median abdominal scar was well healed with no tendency to keloid formation.

There was partial absence of the distal skin creases of the terminal interphalangeal joints of the fingers, most marked in the index finger.

The nails.—These exhibited outstanding features. Both thumb nails were completely absent, the nails of the indices were small, markedly convex and very hard. The nails of the other fingers became more normal as the little finger was approached, but both the nails of the middle and ring fingers were harder than normal. Apart from onychogryphosis of the left great toe nail, the nails of the toes were normal.

Comment

The symmetry of the nail anomalies is striking. The severity of nail involvement is however, variable. At one end of the scale an affected individual may have only a crack or a ridge on the thumb nails, while at the other end of the scale, marked anomalies of all nails may be present. The thumb nail is always affected most severely and the nail of the little finger least of all.

The fingers.—There was a slight flexion deformity of the terminal joints. Both proximal interphalangeal and distal interphalangeal joints could be hyperextended. The indices were nearly as long as the middle fingers, and the thumbs were short and rotated into the palm of the hands. Both little fingers were crooked.

The hair.—The hair of the scalp was of good texture and abundant; the neck hair-line was low. There was complete absence of axillary hair, but the pubic hair was normal.

Comment

There has been no previous reference to any hair abnormality in this syndrome.

The teeth.—Those remaining showed no significant abnormality.

The ears.—There was a moderate degree of deafness due to a combination of bone and nerve deficit.

The eyes.—Both were slightly myopic. The iris of both eyes showed a dark irregular central portion surrounded by a paler peri-



Fig. 5a.—Roentgenograms of the pelvis show the faint outlines of the iliac horns, which are seen well in Figs. 5b and c. In addition the iliac crests are pointed and flare outwards and the ischiopubic rami are wide and thick. There is bilateral coxa vara, the greater trochanters are pointed and the acetabula are deep.

pheral zone. This was considered by Dr. Lois T. Lloyd of the Toronto General Hospital, to be a common normal variant.

COMMENT

The defect of the iris recorded by Lester,[13] the cloverleaf iris, has not been noticed in subsequent cases and is probably not part of the syndrome.

The skull.—No significant abnormality was present.

The neck.—The apparent shortening of the neck was due to a low neck hair-line, high scapulae, and probably some degree of basilar invagination. Cervical movements were normal.

The dorsal and lumbar spines and thorax.—A moderate kyphosis of the dorsal spine and increased lumbar lordosis was present. The chest was flat; the flattening was accentuated by the underdevelopment of the clavicular heads of the pectoralis major. Spinal movements, apart from flexion, were normal. On flexion, the palms of the hands reached the floor; this excess movement probably occurred at the hip joints.

COMMENT

Increased lumbar lordosis has been recorded as a common finding in the syndrome.[8]

The shoulder region.—The shoulders were wide, square and masculine in type. Both scapulae were small and high and the acromion processes were long. The humeral heads were prominent and appeared to lie more anteriorly than usual. Hypermobility was present at both shoulder joints; on internal rotation the tips of the fingers reached the spine of the sixth cervical vertebra.

COMMENT

Changes around the shoulder joints have been frequently reported.[8, 29] The scapula, however, is one of the most variable bones in the body, but the scapula type is commonly inherited.





Figs. 5b and c.—The "iliac horns" are well seen in this view; that on the right is three inches long and that on the left is two and one-half inches long. Comment: "The iliac horns" are striking both on clinical and on roentgenographic examinations. They do not appear to have any useful function and have not been reported in any other vertebrate.

The elbow region, forearms and wrists.—The carrying angle was increased by 15°, the medial epicondyles were prominent; 15° of flexion deformity was present bilaterally. The forearms were held in the midprone position. Supination was absent, but pronation was present from this midprone position through 60°. Both radial heads were subluxed posterolaterally. No significant abnormality of the forearms was noted. At the wrist the radial and ulnar styloids were at the same level. The lower third of the ulna was bowed posteriorly, leading to the prominent ulnar styloid. Movements at the wrist joint appeared normal.

COMMENT

These changes are typical; again they are symmetrical. It has been postulated that the underdevelopment of the capitellum is the primary defect and that the radial head slips off this poorly developed structure. The wrist changes are presumably secondary to the subluxation of the radial heads.

The pelvis.—The typical pelvic changes were seen on the roentgenograms (Fig. 5). The "iliac horns" were readily felt.

The hip joints.—Both joints were hypermobile. Abduction to 90° was present bilaterally.

The knee joints.—The right patella was hypoplastic and dislocated posterolaterally. The left patella was hypoplastic and subluxed laterally. Both medial femoral condyles were prominent. Flexion was full and of normal strength; extension was only slightly impaired in power but the range was normal. There was no instability of the joints.

COMMENT

These are typical features.

The calves, ankles and feet.—The calves were thin and underdeveloped. Ankle and foot movements were normal. The feet were long, thin and slightly flat.

Laboratory investigations.—The hæmoglobin was 12.1 g. %, and the red blood cell count was 4,400,000/c.mm. The sedimentation rate was 9 mm./hour (Westergren). Urinalysis and Wassermann tests were negative. Serum calcium was 9.2 mg. %, serum protein 6.6 g. %, (albumin 5.0 g., globulin 1.6 g., albumin/globulin ratio 3.1:1).

GENETICS

Aschner[1] concluded that there were probably three genes responsible for the



Fig. 6a.—Roentgenograms show the right patella subluxated posterolaterally. The left patella is not well visualized. Both lateral femoral condyles are hypoplastic. Mild osteoarthritis is present.

various abnormalities in this syndrome and that they were closely linked.

Montant and Eggerman[16] concluded that a single gene was responsible for the different abnormalities. This is the concept of polyphenia, which assumes the origin of different abnormalities from a single dominant gene. It is also the assumption of Trauner[28] and Oesterreicher.[18]

The recent work of Renwick,[19, 20] and Lawler *et al.*[10, 11] has thrown further light on the inheritance of this condition. They believe that a single autosomal dominant gene is capable of producing all the abnormalities, but that there are probably modifying influences in the region of the genetic site which causes the different combinations of abnormalities. The chief of these appears to be the particular modifying gene received from the normal parent. They have evidence of a linkage between ABO blood groups and the gene involved in this syndrome. They are unable to state whether the pelvic abnormalities, particularly the presence of the iliac horns, are due to chance, suppression, or to a second variety of a major gene.

In summary therefore, the condition is transmitted by an autosomal dominant gene with pleitropic effects. The expressivity of the syndrome is variable, depending on modifying influences at the genetic site.

Diagnosis

It is probable that many cases of absence, hypoplasia, or dislocation of the patella, or subluxation of the radial head, occur without being recognized as part of this syndrome. Two further cases of subluxation of the radial head have been seen recently at the Hospital for Sick Children, Toronto, and these children are probably carriers of the abnormal gene.

The diagnosis in a typical case, provided the clinician is aware of this syndrome, does not present great difficulty. A nail anomaly, particularly of the thumb nail, combined with a patella or radial head defect, will most likely fall into this cate-





Figs. 6b and c.—Roentgenograms show the lateral views of the knee joints. The right patella is not seen as it has slipped posterolaterally behind the lateral femoral condyle. The hypoplasia of the lateral femoral condyles is well seen.



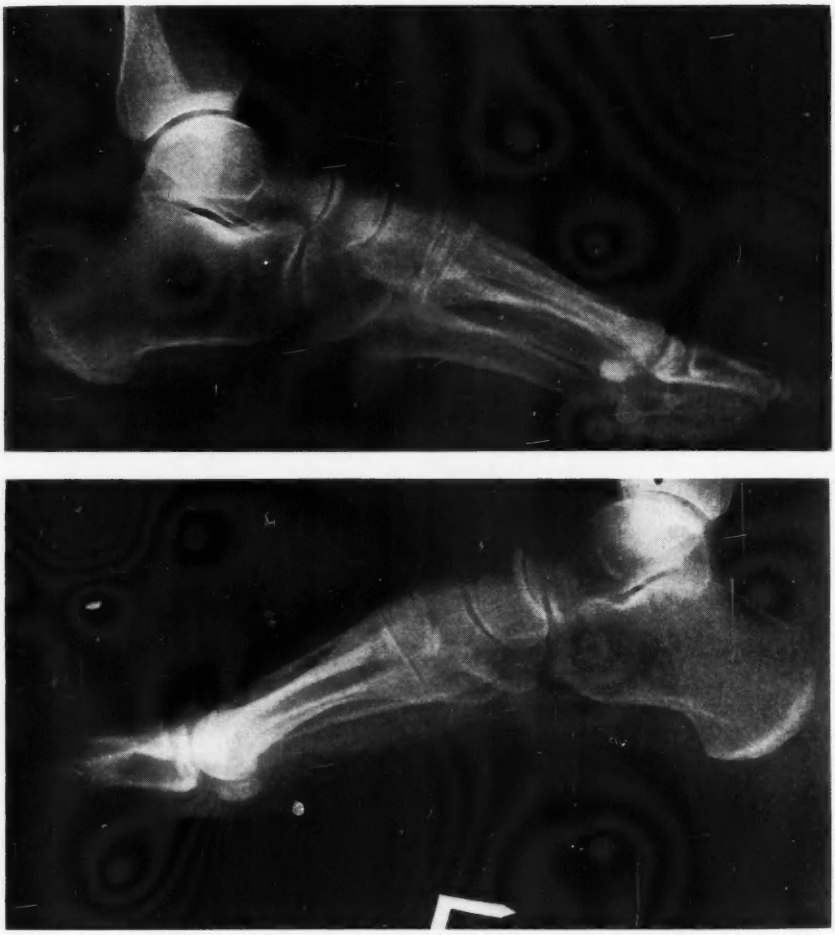
Fig. 6d.—The photograph shows the shape of the knees and the lower extremities. The right patella is dislocated so far laterally and posteriorly that it cannot be seen from this view. The outline of the subluxated left patella can be seen. Both calves are poorly developed and the feet are long and thin. Apart from an onychogryphotic left great toe nail, the nails of the other toes are normal.

gory. In combination with ectodactyly or syndactyly, a nail anomaly without other skeletal manifestations appears to be a separate clinical and genetic entity.[12]

It is apparent that observation of the nails and fingers is still of fundamental importance in diagnosis. Just as the thoracic surgeon or internist may glean important information about the state of the thoracic organs from observation of the nails, so may the orthopædic surgeon glean information regarding the skeletal system.

Orthopædic Problems

The lack of published reports suggests that many of these patients do not seek medical aid.



Figs. 7a and b.—Roentgenograms of the feet do not show any significant abnormality. The superior surface of the os calcis in its posterior third is unusually straight.

Disabilities are related to the absence of the thumb nail, which makes the thumb clumsy, particularly when trying to pick up small objects such as pins. Artificial nails do not appear to help; these have been tried and discarded by the patient whose case history is described.

The subluxed or dislocated patella gives rise to knee instability, with little or no pain. The surgical treatment would depend upon the age of the patient and the associated abnormalities which are commonly found around the knee. It may be necessary to combine a number of procedures, such as tibial tubercle transplantation, plication of the capsule and elevation of the lateral condyle, to correct all the deformities and retain the patella in normal alignment. With the development of secondary osteoarthritic changes, patellectomy may be indicated.

The limited supination resulting from subluxation of the radial head, is minimized, particularly in children, by the accompanying hypermobility of the shoulders and fingers. The changes around the elbow joint, namely the hypoplasia of the capitellum and the hypoplastic subluxed radial head, appear analogous to those seen in the acetabulum and the head of

the femur in congenital dislocation of the hip. Realignment and fixation of the radial head with the capitellum might stimulate normal development in both. If operation is warranted, reduction and fixation of the radial head by a tendon graft passed around the neck of the radius and through the ulna might prove satisfactory. In adults the disability is accentuated by the further changes which have occurred around the inferior radio-ulnar joint. In spite of this, these patients appear to cope so well with their disabilities that operation does not appear indicated.

There are no reports of either "iliac horns" or spinal changes giving rise to symptoms which have required surgical treatment.

Nomenclature

The many anomalies of the skeletal system combined in this syndrome has led to a great variety of titles, as can be seen from the bibliography. The commonest combination, of nail and patella anomalies, has given rise to the title 'The nail-patella syndrome'. The essential entity in the diagnosis is a nail abnormality combined with other skeletal defects, which may or may not include a patella defect. The title 'Hereditary onycho-mesodysplasia' has been suggested as an alternative. It includes the essential nail anomaly, embraces the widespread mesodermal defects which are often present, and accentuates the hereditary nature of the condition.

Summary

The literature on 'The nail-patella syndrome' has been reviewed. The clinical and radiological features have been demonstrated.

The diagnosis, orthopædic problems, genetics and nomenclature of this syndrome have been considered; the title 'Hereditary onycho-mesodysplasia' has been suggested.

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RÉSUMÉ

En 1897, Little[14] releva de la littérature 42 cas d'absence congénitale ou de retard de développement de la rotule, et y ajouta trois autres cas de seins. Dans cet article il mentionne une famille étudiée par William Sedgwick, dans laquelle, à travers quatre générations, 18 membres n'avaient ni rotule ni ongle au pouce. Ceci constitue la plus éloignée à une association de ces deux anomalies.

En 1912, Firth[4] cite une autre famille où les deux difformités étaient soit toutes deux présentes ou toutes deux absentes. Il mentionna en plus que la mère et deux enfants montraient des mouvements de supination de l'avant-bras et d'extension du coude incomplets. Cet article constituait la première référence à une triade associant une anomalie des ongles, des rotules et des coudes.

Turner[51] en 1933 donnait une description très nette du syndrome en mentionnant que plusieurs autres anomalies pouvaient coexister.

Plus récemment, Jameson[8] et Lawler[10, 11] et leurs collaborateurs ont entrepris des études génétiques poussées des familles affublées de ce syndrome.

En résumé, la transmission de cet état se fait par un gène autosome dominant, avec effets pleiotropiques.

La nomenclature a été brouillée par les nombreuses anomalies du système squelettique, associées à ce syndrome. L'entité essentielle du diagnostic étant une anomalie unguéale, associée à d'autres défauts squelettiques, le terme "onychomésodysplasie héréditaire" est suggéré.

A CLINICO-HISTOLOGIC CONSIDERATION OF RENAL MALFORMATIONS*

"The enlarging role of the urologist in medicine makes increasing demands on his investigative skill and diagnostic enterprise. No longer is it sufficient to be a surgeon alone. He must also now be a physiologist, a bacteriologist, and a pathologist. A few years ago nephrectomy and prostatectomy were paramount tests of his skill, but now the intricacies of adrenal gland diagnosis and surgery, operations for urinary diversion, arteriography, and so forth are absorbing his investigative energies. Still challenging him, however, with little or no change apparent in knowledge as to pathogenesis, is the intricate symptom-complex we call pyelonephritis, and this is also true of its first cousin 'hypertension'. . . .

". . . A statistical analysis of combined reports from the world literature, based on gross pathological findings, reveals that in a series of 1,027,904 autopsy reports, combined by the author, congenital absence of one kidney occurs once in 1,285 persons. On the basis of a smaller series, unilateral hypoplasia occurs once in approximately every 600 persons, and double kidney in one out of every 150 people. Therefore, it is apparent that these anomalies occur in a segment of our population sizable enough to be of major importance. Although in the minority, a few articles in the literature on congenital malformations of the kidney carefully consider histological findings in congenitally grossly malformed kidneys, and such relationship to pyelonephritis. . . ."

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PERITALAR DISLOCATION OF THE FOOT

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Dislocations of the talocalcaneal and the talonavicular joints often occur simultaneously and such injuries have usually been referred to in the literature as subastragalar or subtalar dislocation. The authors feel that the term "peritalar dislocation of the foot" more accurately describes this condition. The purpose of this paper is to review the literature on this problem and to report six cases of peritalar dislocation seen locally within a period of 15 months.

It is interesting to note that this condition was described in the pre-roentgen era. According to Bonnin,[1] Broca (1853) classified four types; medial, lateral, anterior and posterior, depending upon the direction of displacement of the foot. Shands[7] in 1928 reported a patient with inward subastragaloid dislocation of the foot, the first case in the records of the Johns Hopkins Hospital. In 1937 Smith[8] reported seven cases of subastragaloid dislocation found among 535 patients with traumatic dislocations of all types seen at the Campbell Clinic, Memphis, Tennessee. He felt that this injury was not as rare as the literature suggested. Leitner[3] in 1952 reviewed the case reports of 4125 patients with traumatic dislocations seen at Boehler's Clinic, Unfallkrankenhaus, Vienna, from 1925 to 1950, and of these 42 are subtalar. In this series of dislocations, 36 were medial, five were lateral and one posterior. Leitner[4] in a later report described the obstacles to closed reduction in four patients with subtalar dislocation. In 1953 Mulroy[5] described a patient with lateral dislocation which required open reduction to disengage the posterior tibial tendon from the lateral side of the neck of the talus. Plewes and McKelvey[6] in 1944 noted that avascular necrosis of the body of the talus does not occur after subtalar dislocation.

Case Reports

Case 1.—The patient was a 54-year-old woman who injured her left foot in an automobile accident on July 2, 1958. She was examined two days later, on July 4. There was a pronation deformity of the left foot with a blister one inch in diameter over the medial side of the head of the talus. Radiographs showed a lateral peritalar dislocation of the foot with a bimalleolar fracture of the ankle, and fractures of the bases of the second, third and fourth metatarsals (Figs. 1a and b). On July 4, 1958, under general anæsthesia, closed reduction was performed without difficulty and a cast was applied. Open reduction and screw fixation of the medial malleolus was performed on July 31, 1958, and a patch of dry gangrenous skin, one-half inch in diameter over the medial side of the head of the talus was excised and the wound closed primarily. Full weight-bearing was permitted on October 18, 1959. At recheck examination on April 25, 1960, 22 months after injury, the patient complained of a mild aching in the left foot and there was a grade 2 limitation of inversion-eversion. Radiographs showed hypertrophic changes at the talonavicular joint and there was no evidence of avascular necrosis of the talus.

Case 2.—A 46-year-old woman who was first seen on September 5, 1958, had a chief complaint of pain in her left foot and ankle on walking. Her left foot was injured in an automobile accident on September 26, 1957. On examination there was a slight varus deformity of the foot with lack of the normal heel prominence, and a painful grade 3 limitation of inversion-eversion motion of the foot. Radiographs taken on the day of injury showed a fracture of the lateral malleolus, with an anterior peritalar subluxation of the foot and a fracture of the lateral process of the body of the talus (Fig. 2a). Radiographs taken on September 5, 1958, at the time of examination, showed persistent anterior subluxation of the foot with healing of the fractures. Triple arthrodesis was recommended but the patient refused operation. On May 5, 1960, 32 months after the injury she stated that the pain was less severe, and there was a grade 4 limitation of inversion-eversion of the foot. Radiographs showed an apparent talocalcaneal fusion, with persistent widening and degenerative changes at the talonavicular joint (Fig. 2b).

Case 3.—The patient was a 21-year-old man who injured his left foot on December 19, 1958, when the motorcycle he was riding



Fig. 1a.



Figs. 1a and b.—(Case 1). Lateral peritalar dislocation of the foot. The lateral radiograph shows slight overlapping of the talus and navicular, indicating some posterior displacement of the foot. The supero-inferior view shows the head of the talus medial to the cup of the navicular, indicating lateral displacement of the foot.

was in collision with an automobile. He was seen shortly after the accident. There was a marked pronation deformity of the left foot. There was cyanosis, hypoæsthesia and inability to move his toes. Radiographs showed a lateral peritalar dislocation of the foot, with a chip fracture of the lateral process of the body of the talus. Immediate closed reduction was accomplished without difficulty under spinal anæsthesia, and a cast was applied. Motion, sensation and circulation of the toes returned to normal. Full weight-bearing was allowed on March 13, 1959. On recheck examination on April 9, 1960, 16 months after the injury, the patient complained of a mild aching in the left foot. There was a grade 2 limitation of inversion-eversion, and a firm nodule, 1 cm. in diameter, postero-inferior to the medial malleolus was palpated. Pressure over the nodule resulted in radiating pain into the sole of the foot and toes. The nodule was thought to be a traumatic neuroma of the posterior tibial nerve. Radiographs showed hypertrophic changes at the talonavicular joint and there was no evidence of avascular necrosis of the talus.

Case 4.—This patient was a 42-year-old man who injured his left foot on October 18, 1959, when his left trouser leg was caught in the power take-off of a corn picker. He was examined shortly after the accident. There was a marked pronation deformity of the foot. Radiographs showed a lateral peritalar dislocation of the foot and a chip fracture of the lateral malleolus. Under general anæsthesia, immediate closed reduction was accomplished without difficulty and a cast was applied. Full weight-bearing was allowed on December 1, 1959. The patient was examined on March 18, 1960, five months after the injury. He complained of mild aching and there was a grade 1 limitation of inversion-eversion of the foot. Radiographs showed no evidence of avascular necrosis of the talus.

Case 5.—A 49-year-old man was injured in an automobile accident on October 21, 1959. He was lying under his automobile making repairs when the hoist gave way and the car fell on him. He was seen shortly after the accident. He sustained multiple injuries including compression fractures of three lumbar vertebrae, a comminuted fracture of the left femur and an injury to the left foot. There was a marked adduction deformity of the foot and radiographs showed a medial peritalar dislocation. Under general anæsthesia, immediate closed reduction of the foot was



Figs. 2a and b.—(Case 2). Anterior peritalar dislocation of the foot. The lateral radiograph, taken on the day of the injury, shows widening of the talo-navicular joint, with forward displacement of the os calcis under the talus. The supero-inferior view, taken 32 months after the injury. shows persistent widening of the talo-navicular joint with degenerative changes medially. (A supero-inferior view was not taken at the time of injury.)

accomplished without difficulty and a cast was applied. Weight-bearing has not been permitted because of the fractured femur which was slow to unite. On April 21, 1960, six months after the injury the patient did not have pain in the foot and there was a grade 3 limitation of inversion-eversion of the foot. Radiographs showed marked osteoporosis of the foot and there was no evidence of avascular necrosis of the talus.

Case 6.—The patient was a 36-year-old woman who received multiple injuries in an automobile accident on August 15, 1959. She was treated elsewhere and was first seen by the authors on January 28, 1960. Radiographs taken on the day of the injury showed a compression fracture of the 10th thoracic vertebra, a fracture dislocation of the right hip and a medial peritalar dislocation of the left foot with a fracture of the posterior process of the body of the talus (Figs. 3a and b). The fracture dislocation of the right hip was treated by closed reduction and plaster fixation. Initial closed reduction of the left foot was unsuccessful, but closed reduction was accomplished on August 25, 1959, and a cast was applied. On May 20, 1960, nine months after the injury the patient did not complain of pain and there was a grade 4 limitation of inversion-eversion of the left foot. Radiographs showed bony union between the posterior process of the body of the talus and the calcaneus. There was no evidence of avascular necrosis of the talus.

Discussion

The authors agree with Smith that this injury is more common than the literature would indicate. Table I represents a summary of our six cases. As might be expected there was no age or sex preponderance. Peritalar dislocation is produced by





Figs. 3a and b.—(Case 6). Medial peritalar dislocation of the foot. The lateral radiograph shows minimal overlapping of the talus and navicular, as well as a fracture of the posterior process of the body of the talus. The supero-inferior view shows the head of the talus lateral to the cup of the navicular, indicating medial displacement of the foot.

Fig. 3b.

severe torsional trauma to the foot. Its presence should be expected whenever there is a marked varus or valgus deformity of the foot following injury. Active motions of the foot are limited and painful and there may be evidence of circulatory insufficiency in the toes. There is increased tension of the skin over the side of the foot and ankle opposite to the direction of the displacement and if reduction is delayed, blistering and subsequent necrosis of the skin may occur. The diagnosis is confirmed by radiography. The changes in the lateral view may be subtle and misleading in some instances. We do not agree with Watson-Jones[9] who emphasized the equinus position of the talus as seen in the lateral roentgenogram; the talus was not in equinus in any of our patients. We feel that the superior-inferior radiograph of the foot is most important, and one should look for displacement of the head of the talus from the "cup" of the navicular. Usually displacement of the foot occurs in two planes, there being an associated forward or backward displacement of the foot when the dislocation is medial or lateral. We have classified our cases according to the major displacement. The dislocation was lateral in three patients, medial in two patients and anterior in one patient. Fractures of the foot and ankle frequently accompany peritalar dislocation of the foot. Three patients had fractures involving the ankle mortise and three patients had fractures of the talus (Table II).

Peritalar dislocation can usually be reduced without difficulty by manipulation which should be performed immediately the diagnosis is made. Obstacles to reduction were not encountered in any of our patients. One patient (Case 6) required a second closed reduction while in another (Case 2) the dislocation was apparently unrecognized and reduction was not attempted. If closed reduction is unsucces-

TABLE I.—PERITALAR DISLOCATION OF THE FOOT: CASE SUMMARIES

Case No.	Age in years	Sex	Type of accident	Side of dislocation	Direction of dislocation
1	54	F	automobile	L	lateral
2	46	F	automobile	L	anterior
3	21	M	motorcycle	L	lateral
4	42	M	trouser leg caught in power take-off	L	lateral
5	49	M	automobile (hoist)	L	medial
6	36	F	automobile	L	medial

TABLE II.—PERITALAR DISLOCATION OF THE FOOT: ASSOCIATED FRACTURES OF THE FOOT AND ANKLE

Case No	
1	Bimalleolar fracture and fractures of 2nd, 3rd and 4th metatarsals.
2	Lateral malleolus and lateral process of body of talus.
3	Lateral process of body of talus.
4	Chip fracture of lateral malleolus.
5	None.
6	Posterior process of body of talus.

TABLE III.—PERITALAR DISLOCATION OF THE FOOT: FOLLOW-UP

Case No.	Length of follow-up months	Pain	Limitation of inversion-eversion	Radiographic findings
1	22	minimal	grade 2	talo-navicular arthritis
2	32	moderate	grade 4	talo-calcaneal fusion
3	16	minimal	grade 2	talo-navicular arthritis
4	5	minimal	grade 2	unremarkable
5	6	unassessed	grade 3	osteoporosis
6	9	minimal	grade 4	talo-calcaneal fusion

ful, operation should be undertaken without delay in order to remove obstructing tissues under direct vision. Weight-bearing should be withheld for six to eight weeks to allow for soft tissue healing.

Some of the findings that were noted at the follow-up examinations are presented in Table III. The length of follow-up varied from five to 32 months. All patients who were walking on the injured foot complained of pain but usually it was mild and was not a problem. Limitation of inversion-eversion motion of the foot was present in all patients, being partial in four patients and complete in two. The latter two patients had fractures of the body of the talus and there was apparent bony union between the talus and os calcis on the radiographs. Avascular necrosis of the body of the talus did not occur in any of our patients. Haliburton *et al.*[2] have shown that the body of the talus is mainly supplied with blood by vessels which enter antero-inferiorly through the roof of the tarsal canal, and by vessels which enter the medial surface below the articular facet for the medial malleolus. In peritalar dislocation of the foot, the vessels entering the body of the talus from the tarsal canal are likely interrupted, while those entering medially probably survive and are sufficient to maintain the viability of the bone.

CONCLUSIONS

Peritalar dislocation of the foot is a more common injury than the literature would indicate.

Medial and lateral dislocations are the most common, while anterior and posterior dislocations are rare.

One should look for displacement of the head of the talus from the "cup" of the navicular on the supero-inferior radiograph of the foot to establish the diagnosis.

Fractures of the foot and ankle frequently accompany peritalar dislocation.

Closed reduction can usually be performed without difficulty. Residual limitation of inversion-eversion movement of the foot is to be expected. Avascular necrosis of the talus rarely, if ever, follows peritalar dislocation.

ACKNOWLEDGMENT

The authors wish to thank Dr. R. C. Rider, Windsor, Ontario, who treated Case 2: Dr. R. K. Lyon, Leamington, Ontario, who treated Case 4, and Dr. C. M. Hamilton, Vernon, B.C., who treated Case 6, for their kind permission to include reports of their patients.

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RÉSUMÉ

Parceque les luxations astragalo-calcanéenes et astragalo-scaphoïdiennes sont souvent associées, les auteurs préfèrent le terme "péritalaire" qui est plus discriptif.

La Table I représente un sommaire de nos six cas. La luxation péritalaire est produite par la torsion brutale du pied. Sa présence devrait être suspectée chaque fois qu'il se manifeste une difformité en varus ou en valgus après un accident.

La luxation péritalaire du pied est plus fréquente que la littérature le laisse croire. La luxation médiale et latérale sont les plus fréquentes, l'antérieure et la postérieure étant exceptionnelles. On se doit de rechercher le déplacement de la tête de l'astragale de la face postérieure du scaphoïde sur les radiographies. Les luxations sont souvent accompagnées de fractures du pied et de la cheville.

La correction se fait habituellement par réduction fermée, et sera suivie d'une limitation résiduelle du mouvement d'inversion-éversion. La nécrose avasculaire de l'astragale est à peu près inconnue consécutivement à la luxation péritalaire.

CHOLELITHIASIS ASSOCIATED WITH CONGENITAL MALFORMATION ON THE CYSTIC DUCT IN A YOUNG CHILD*

"Mechanical obstruction of the biliary system has not been adequately emphasized as a cause of cholelithiasis in children. Since the work of Potter (1928), many authors have enumerated the ætiological factors involved, and though they have included obstruction, they have often failed to elaborate on this detail and to illustrate their comments with clinical examples.

"Cholelithiasis is much less common in children than in adults: Wilenius (1951) found it incidentally in 0.28 per cent of cases at autopsy in the age group from birth to 15 years, compared with Lieber's (1952) figure of 11.6 per cent in adult autopsies in subjects over 20 years of age. In children, there is no marked sex preponderance (Potter, 1938, Ulin, Nosal, and Martin, 1952). DeCamp, Ochsner, Baffes, Bancroft, and Bendel (1952) found the condition to be twice as prevalent in Caucasians as in negroes. Most authorities agree with Potter (1938) that the incidence increases with age within the age group from birth to 15 years. Despite the relative infrequency in children, it has been rightly stressed by Potter (1928) and others that cholelithiasis is a disease which is sufficiently common to merit inclusion in the differential diagnosis of abdominal pain at all ages.

"In view of the fact that cholelithiasis is not often associated with cholecystitis in children, it is surprising that these two distinct conditions have been so inseparably combined in the writings of many authors. Ulin, Nosal, and Martin (1952) state that cholelithiasis occurs only in 57 per cent of cases of cholecystitis in children, as compared with 80-95 per cent in adults. According to Gross (1953), acute cholecystitis in children, the common type of gall-bladder infection in these subjects, is not as common as cholelithiasis. These statements imply that a large proportion of cases of cholelithiasis in children have no accompanying cholecystitis. It would seem that the dissociation of cholelithiasis and cholecystitis in children should be stressed.

"The three generally accepted ætiological factors have been stated by Ulin, Nosal, and Martin (1952) to be infection, disordered metabolism, and disordered function (including obstruction). Infection has been ably discussed by many writers, who remark on the more fulminating process which occurs in a child usually as an infection in the gall-bladder secondary to infection elsewhere in the body. A large number of conditions have been incriminated as the primary source of infection. Some of these are no longer prominent, as in the case of typhoid fever, and others have not withstood the test of intensive investigation, such as scarlet fever (Swing and Bullowa, 1938). Nevertheless, salmonella infection of the intestine, appendicitis as a primary source of metastatic spread to the gall-bladder, and lambliasis infestation (Lowenburg and Mitchell, 1938) are still held responsible among other conditions for providing an infective factor which may predispose to gall-stone formation. However, as Sallick (1943) has mentioned, most acute inflammations of the gall-bladder in children subside spontaneously, and they are rarely complicated by chronic cholecystitis or the formation of stones. . . ."

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*WALKER, G. R., FIELD, P. AND CONEN, P. E.: *Brit. J. Surg.*, **48:** 78, 1960.

INTESTINAL OBSTRUCTION DUE TO LANE'S ILEAL KINK*

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THIS is a report of a rare cause of small bowel obstruction.

Paul and Fernando[1] recently directed attention to a lesion which they call "Lane's ileal kink" and pointed out the difference between the pathological findings in their cases and the findings described originally by Lane. They have described two cases of intestinal obstruction caused by such a kink and indicated that these were the only such cases in the literature.

Lane[2-6] postulated that when man assumed the erect posture, a loaded cæcum pulled on the terminal ileum; this in turn caused a thickening in the posterior layer of the mesentery of this part of the bowel, particularly at the junction of the part of the ileum possessing a complete mesentery and the small distal segment of the ileum in the ileocæcal angle where the posterior layer of the mesentery is absent. In time, this stress was alleged to cause thickening in the posterior lamina of the mesentery. Although this primarily supported the ileum in spite of prolapse of the cæcum, the band could eventually cause a kink in the ileum, resulting in ileal stasis which Lane ascribed to this mechanism.

The lesions, as described in Paul and Fernando's two cases, were likewise non-inflammatory peritoneal thickenings which extended from the antimesenteric border of the terminal ileum to the posterior abdominal wall in relation to the rim of the pelvis. This fixation of the terminal ileum could only be released by operation, and when this was done, no thickening in the posterior layer of the mesentery was discovered. However, they suggested that the term "Lane's ileal kink" be used for the lesion they described although it differed in detail from that originally described by Lane. The case reported here presented as a small bowel obstruction and the findings at operation were as described by Paul and Fernando.

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Case Report

Mr. J.L., aged 69 years, was admitted to the University Hospital, Saskatoon, with a four day history of crampy lower abdominal pain associated with continued vomiting and inability to pass flatus or stool. He had been given intravenous fluid before his admission to hospital. Several enemas had produced no relief of the obstruction.

Previous history.—He had enjoyed good health for many years. Peptic ulcer symptoms which he had had since his youth were relieved after partial gastrectomy in 1949. He had asymptomatic bilateral inguinal herniae of many years' duration. He had always been able to reduce both herniae.

Examination.—The patient was a dehydrated elderly man, with a normal blood pressure and a subnormal temperature. The chest and cardiovascular system revealed no abnormality on clinical examination. The blood pressure in the upper extremity was 140/90, on admission.

The abdomen was moderately distended and tympanitic throughout. No visible peristalsis was observed. There was no muscle guarding or rebound tenderness and no evidence of free fluid. The liver, spleen and kidneys were not palpable. There were no palpable masses. Rectal examination was unremarkable and the bilateral direct herniae were painless, soft and easily reducible. Bowel sounds were audible over the entire abdomen; their character in the right lower quadrant was abnormally high-pitched and tinkling.

Radiological examination. — Chest radiographs taken on admission showed no abnormal findings. Films of the abdomen revealed low small bowel obstruction (Fig. 1).

Laboratory findings on admission.—He had a pronounced leukocytosis. The total white blood count was 50,050/c.mm. with 36% band forms. Blood electrolytes on admission were: sodium 129 mEq./l., chloride 93.1 mEq./l., bicarbonate 22.2 mEq./l., potassium 4.75 mEq./l. Non-protein nitrogen was 89 mg. %.

After gastric suction, and fluid and electrolyte replacement therapy, his general condition improved. Further films a few hours after admission indicated that the distension of small bowel loops was increasing. Enemas produced no results. Accordingly he was subjected to operation under general anæsthesia,

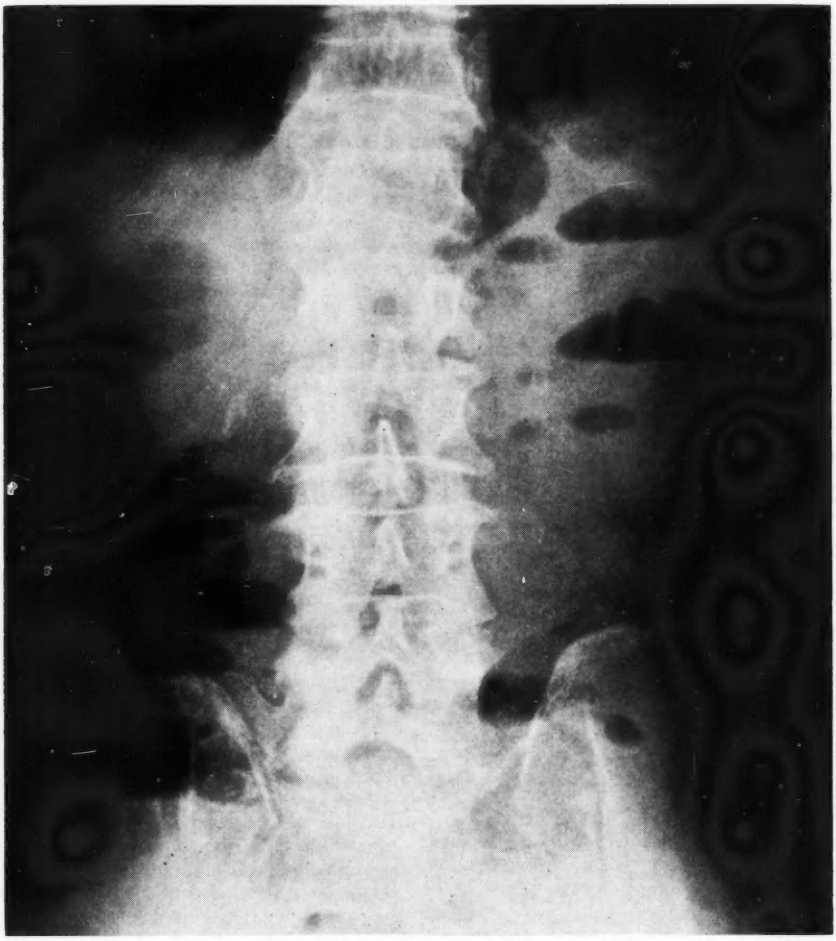
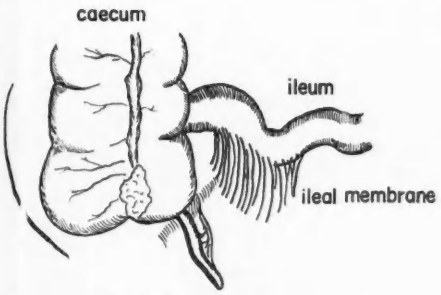


Fig. 1.—Plain upright film of the abdomen showing air and fluid levels in the small bowel.

and the abdomen was opened through a right paramedian incision. Exploration of the abdomen showed a slight increase in the amount of fluid present in the peritoneal cavity; this was clear, serous and sterile on culture. There were no adhesions throughout the abdomen, and the site of the previous gastrectomy appeared to be normal. The lower small bowel was distended to a moderate degree, proximal to a kinked portion located some four inches from the ileocæcal valve. The cæcum and colon were collapsed and empty. There were no adhesions in the area of the appendix and the appendix appeared perfectly normal. The terminal ileum was adherent to the posterior abdominal wall in the cases as described by Paul and Fernando. Division of the peritoneal band (Fig. 2) at once released this segment of ileum and peristaltic activity immediately recommenced. A rather pultaceous mass which had been arrested at the junction of the dilated and constricted segments of the ileum was milked through into the cæcum. This procedure took only a short while and the bilateral herniae were then repaired by Henry's method, after closing the peritoneum. The subsequent course in hospital was uneventful except for some fever on the second and third postoperative days. Radiographs of the chest at this time showed some infiltration in the right lower lobe. The fever and radiological changes rapidly cleared with physiotherapy and antibiotics.



LANE'S ILEAL MEMBRANE.

Fig. 2.—Drawing to show ileal membrane extending from the antimesenteric border of the ileum to the pelvic brim.

Conclusions

This case of intestinal obstruction appeared to be caused by Lane's ileal kink as documented by Paul and Fernando. This pathological fixation of terminal ileum with subsequent kinking of the bowel appeared to be due to a congenital band. There was no evidence of postinflammatory adhesions; the peritoneum itself appeared to be healthy as did the mesenteric lymph nodes.

Careful examination of the stools was carried out after operation. No evidence of a vegetable bezoar was found; the precipitating cause of the obstruction was believed to be undigested food residue which had become impacted proximal to the ileal kink.

Our purpose in reporting this case is to draw attention to this rare cause of intestinal obstruction which has only been reported in two previous cases in the literature.

Acknowledgment

We wish to thank Dr. C. L. N. Robinson for permission to publish this case history.

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Résumé

Paul et Fernando[1] ont attiré récemment l'attention sur une lésion qu'ils appellent la "Boucle iléale de Lane" et ils ont illustré les différentes observations originales décrites par Lane, en deux cas d'obstruction intestinale causée par une telle boucle.

Le cas rapporté ici présentait un tableau clinique et radiologique d'une obstruction iléale basse. Les observations per-opératoires furent identiques à celles déjà décrites par Paul et Fernando: un iléon distendu modérément en amont d'une portion iléale bouclée, située à quelques quatre pouces de la valvule iléocœcale et fixée postérieurement à la paroi abdominale. La division de la lame péritoneale (Fig. 2) mobilisa immédiatement ce segment d'intestin qui reprit tout de suite son péristalisme normal. Ce cas semble être le troisième qu'on ait rapporté, où il s'agisse d'une "Boucle iléale de Lane" avec phénomènes d'obstruction précipités par une accumulation de détritus alimentaires.

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COLOCYSTOPLASTY FOR BLADDER SUBSTITUTION: REPORT ON 45 CASES

J.-P. BOURQUE, F.R.C.S.[C], F.A.C.S., F.I.C.S.,* *Montreal*

We have always been extremely interested in the problem of bladder enlargement and substitution. With Couvelaire and other pioneers in this field, we have utilized the ileum in this type of surgery in the past; but for the past three and one-half years, that is since early 1957, with René Kuss, J. M. Gil Vernet and Goodwin, we have adopted the sigmoid colon for bladder enlargement and substitution for the following reasons: (1) The sigmoid colon is already a large reservoir, (2) the sigmoid is already a low pelvic organ, (3) this intestinal segment is easily brought down in the retropubic space and can always be placed in iso-peristaltism, (4) the newly formed sigmoid-bladder can be completely extra-peritonealized and thus all the anastomoses made outside of the peritoneal cavity; this fact alone gives a much greater postoperative security, (5) the mesentery of the sigmoid, in contrast to that of the ileum, does not partition the abdominal cavity, and for this reason the risks of intestinal subocclusion or occlusion in the immediate or late postoperative course are much diminished, (6) the ureters are easily brought down to the new-formed bladder for anastomosis without displacing them much from their normal situation. This avoidance of long dissection salvages the blood supply of the ureters, so the healing at the site of the anastomosis is of a much better quality because the risks of stenosis that could compromise renal vitality are much diminished, (7) the expulsive muscular system of the sigmoid colon is superior to that of the ileum, (8) this intestinal segment secretes less mucus than any of the other segments, and thus there is less risk of urinary retention as has been observed with the use of the ileum, (9) being away from the active digestive zone, this segment resorbs much less than the cæcum or the ileum so electrolyte disturbances are exceptional, (10) this reservoir is naturally large so we can hope for a greater bladder capacity with, naturally, much less urinary frequency.

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Indications

We have performed colocystoplasties for enlargement and substitution of the bladder in: (1) The small contracted tuberculous bladder, (2) cases of inveterate interstitial cystitis with or without Hunner's ulcer, where all other procedures had failed, (3) chronic sclerosing or retractile cystitis, (4) operable cancers of the bladder.

According to the different types of pathology, the level of sections made at the cystectomy stage has been different; (a) in the small contracted bladder we retain the bladder neck only and remove all the rest of the bladder; (b) in interstitial and retractile cystitis we preserve the bladder neck and the trigone; (c) in cancer of the bladder we are very radical as after iliac lymphadenectomy we remove bladder, prostate and seminal vesicles making the anastomosis of the new-formed bladder either to the beak of the prostatic capsule or to the membranous urethra, inside the external sphincter (Fig. 1).

Preoperative Care

First, and above all, we have to make sure that the urethra is of normal calibre and that the bladder neck has good patency; if the latter is thick, strictured or sclerosed, it is better to resect it before or during the operation.

We then have a barium enema performed to study the material that will have to be utilized in the operation.

We have to make a very elaborate systemic study of the patient, paying special attention to the renal function and to the electrolytes that must be rectified if necessary and possible; an adequate medico-surgical collaboration is thus essential in the majority of cases.

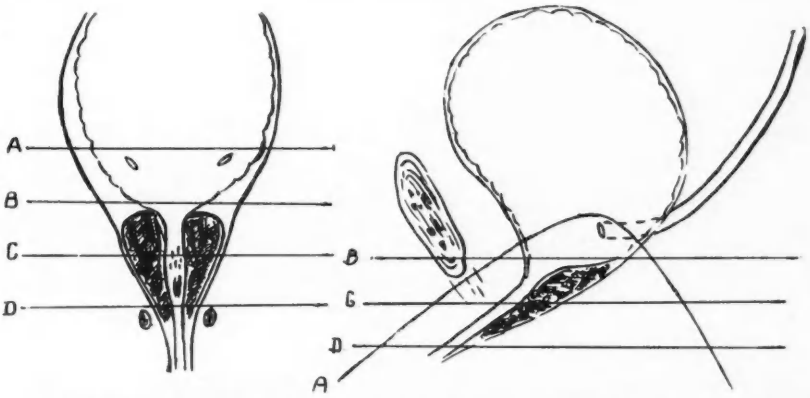


Fig. 1.—Inferior level of subtotal and total cystectomies: (A) In interstitial and retractile cystitis: (B) In the small contracted tuberculous bladder: (C) In cancer of the bladder situated above the trigone: (D) In cancer of the bladder situated below the trigone and at the bladder neck. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

We have to be assured of a sufficient reserve of blood to be able to make adequate replacements of blood losses during and after the operation.

For the intestinal preparation we use kanamycin *per os*, accompanied by a non-residue diet. It is necessary to give one or two evacuating enemas an hour before the operation, and complete the intestinal preparation by a rectosigmoid irrigation with a 1% neomycin solution.

It is preferable to install a Cantor or Levin tube before the operation, so that the patient will not be distressed by gastro-intestinal distension in the immediate post-operative period.

In most cases we use general anæsthesia with intubation.

Technical Operating Procedure

Our first three operations involved anastomosis by the end-to-end method, but these patients have encountered some micturition difficulties due, we believe, to the segmental contractions of the sigmoid. For this reason we have now adopted the side-to-end method which avoids the aforementioned drawbacks and has given very encouraging results.

We will therefore describe only this last method which is the one we are now using in all our cases (Figs. 2 and 3).

1. *A suprapubic* incision is made from the pubis to five or six centimeters above the umbilicus in order to provide good exposure, and to facilitate the ease of operation.

2. After the peritoneum is opened and the self-retaining retractor is placed, we follow with the exploration of the sigmoid colon, so as to choose very carefully the segment which will be utilized, appreciate its length, see that it goes down easily to the lower pelvis, and ascertain whether it will be possible to perform the anastomoses without traction or tension at any point.

3. We then place the intestinal resection clamps at the chosen levels and isolate the

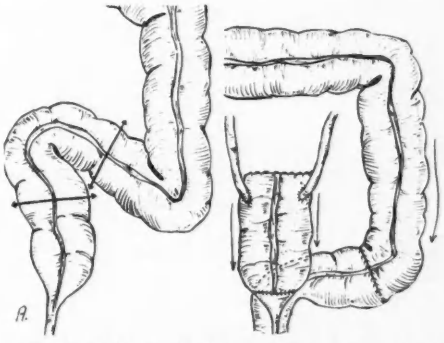


Fig. 2.—End-to-end colocystoplasty. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

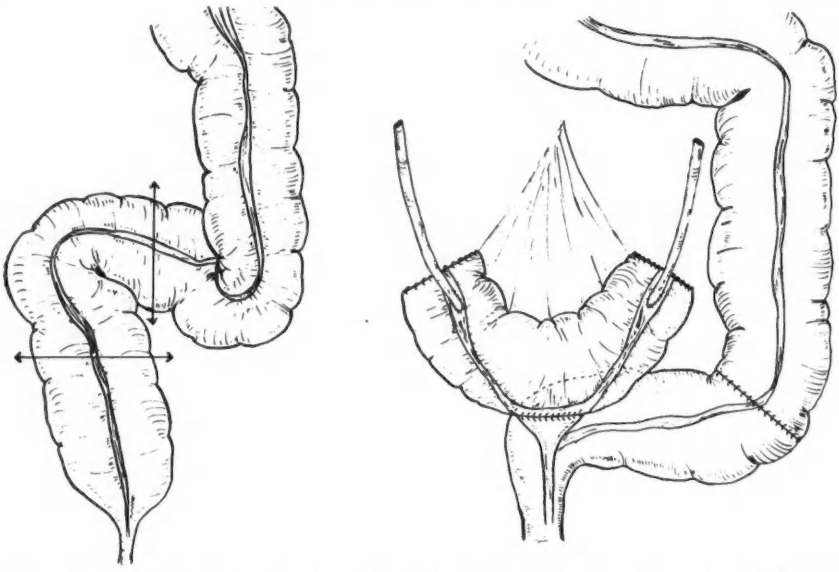


Fig. 3.—Side-to-end colocystoplasty which is the procedure we have adopted. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

segment that will be utilized. We never cut into the mesentery of the sigmoid colon; in this way we ensure that there is no trauma to its vascular system. In this manner, the blood vessels of this portion of the mesentery which will go down into the lower pelvis, merely describe a loop which in no way hampers the blood circulation, and the segment retains a normal colour due to its adequate blood supply.

4. We immediately close the two extremities of the segment in two layers, using 00 silk on the seromuscular layers and 0000 atraumatic chromic catgut on the mucosal layers; we then re-establish the intestinal continuity by an end-to-end anastomosis also in two layers.

5. We then perform a total or subtotal cystectomy, according to the indications of the individual case, by the usual techniques, paying special attention to hæmostasis. Insofar as possible we protect and keep intact the vesical peritoneum which will be used at the end of the operation to extraperitonealize the newly-formed bladder. This peritoneum is incised in the median line down to Douglas' cul-de-sac to provide right and left flaps.

6. If the colonic segment does not go down easily to the bladder neck or to the urethra, it is easy to lengthen its mesentery by sectioning its anterior and posterior parietal sheets being very careful not to injure the blood vessels; this maneuver allows us to gain from 2 cm. to 8 cm. in

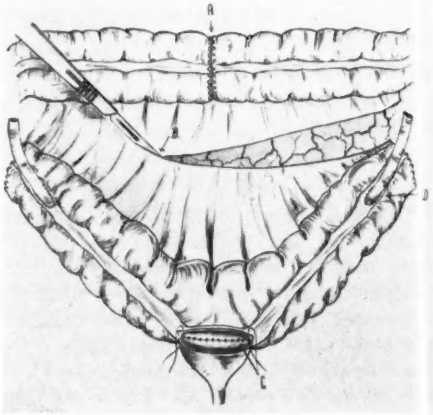


Fig. 4.—(A) Re-establishment of the intestinal continuity has been made: (B) Lengthening the mesentery of the newly-formed bladder: (C) Colo-vesical, cervical, prostatic or ureteral anastomosis being made: (D) Uretero-colic anastomosis.

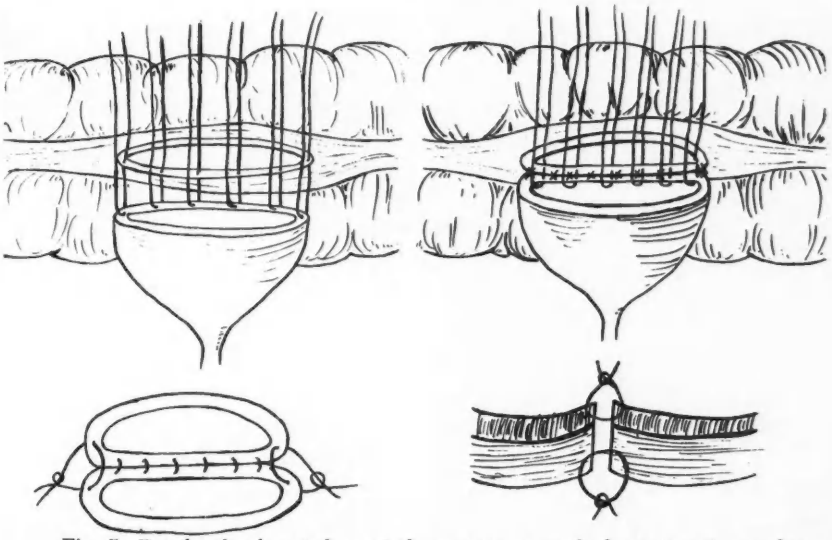


Fig. 5.—Details of colovesical, cervical, prostatic or urethral anastomosis are shown. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

length and permits us to place the segment easily in a side-to-end position even down to the membranous urethra.

7. A longitudinal incision is made on the tænia of the segment, through the sero-muscular layers and then a small patch of intestinal mucosa is resected so as to avoid a mucosal eversion often responsible for the formation of a fistula. The length of this incision will be proportional to the calibre of the trigone, the bladder neck, the prostatic capsule or the urethra, whether

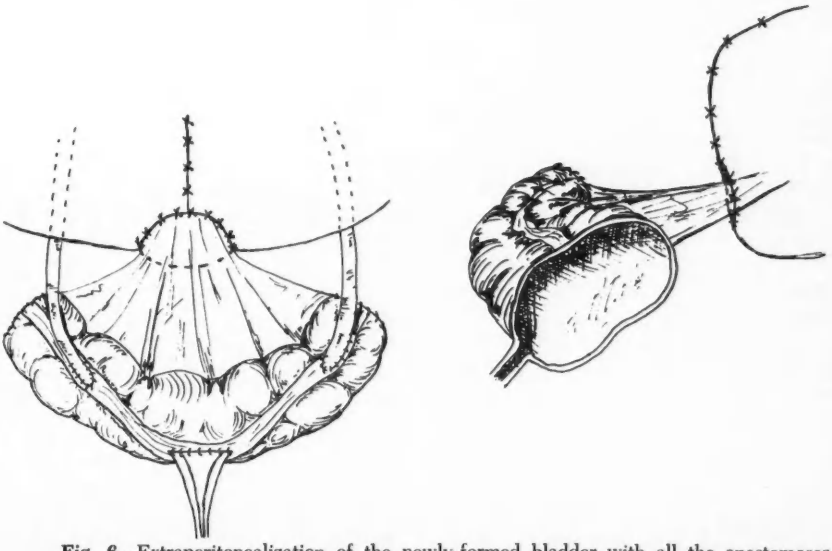


Fig. 6.—Extraperitonealization of the newly-formed bladder with all the anastomoses outside the peritoneal cavity. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

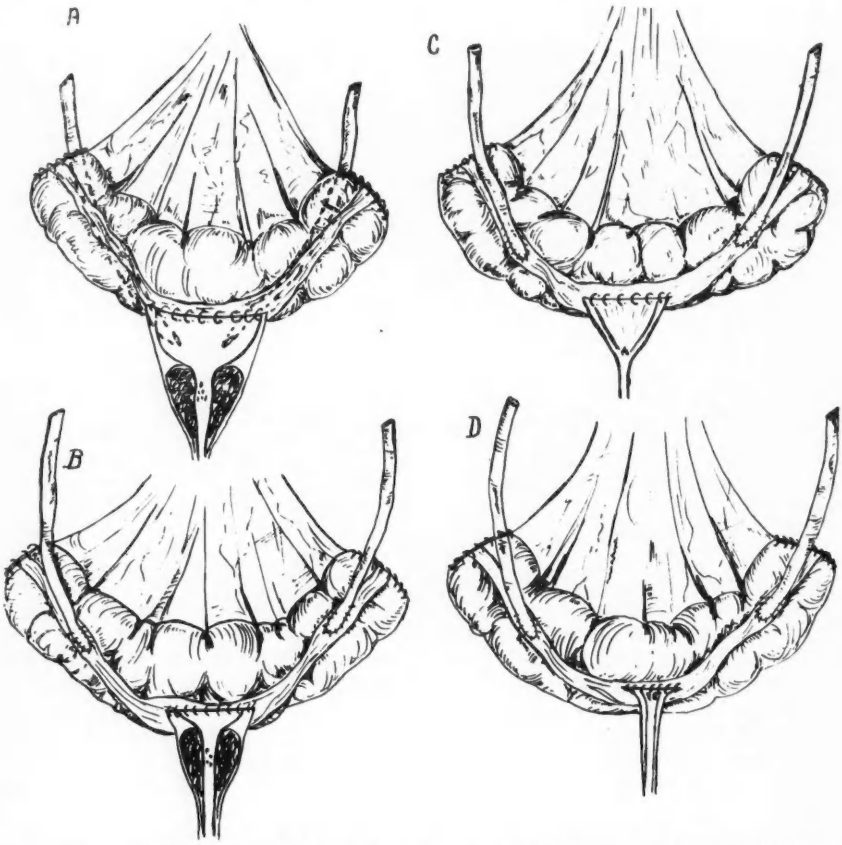


Fig. 7.—(A) Colocystoplasty for interstitial or retractile cystitis: (B) Colocystoplasty for tuberculous contracted bladder: (C) Colocystoplasty for cancer of the bladder situated above the trigone: (D) Colocystoplasty for cancer of the bladder below the trigone or at the bladder neck. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

we perform the anastomosis to one or the other of these anatomical structures.

8. We then proceed with the colo-vesical, cervical, prostatic or urethral anastomosis, according to the indications in the individual case: (a) When the anastomosis is made to the trigone or to the bladder neck we make it in two separate layers, first the posterior seromuscular layer, then the posterior mucosal layer followed by the anterior mucosal layer, and finally the anterior seromuscular layer. The urethral catheter is inserted as soon as the two posterior layers are sutured and is fixed in good position by a silk thread to the urethral meatus in women and to the prepuce in men. We use a *balloonless multiple eye catheter* which provides very efficient drainage: (b) When the anastomosis is made to the urethra it is preferable, in order to avoid the great difficulties which arise from the depth of the anastomosis and the small calibre of the urethra, in suturing the two posterior layers to place the sutures alternately in the muscular and mucosal layers and the anterior layer may be closed in the same manner. In this way we are not confined in a small space which can render the maneuvers very difficult and also compromise the watertight quality required for this anastomosis.

9. We then proceed with the ureterocol c

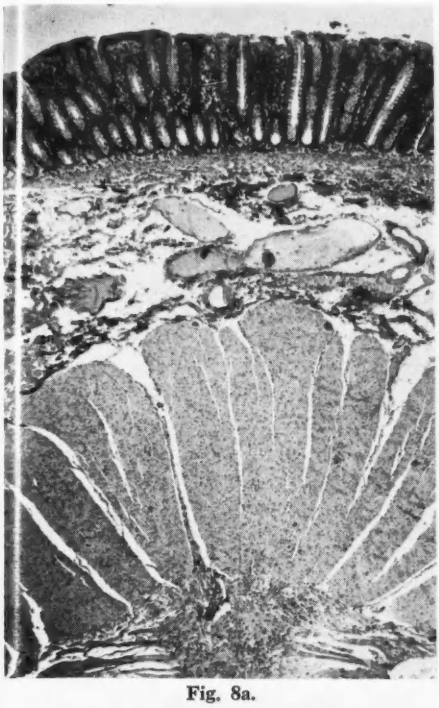


Fig. 8a.

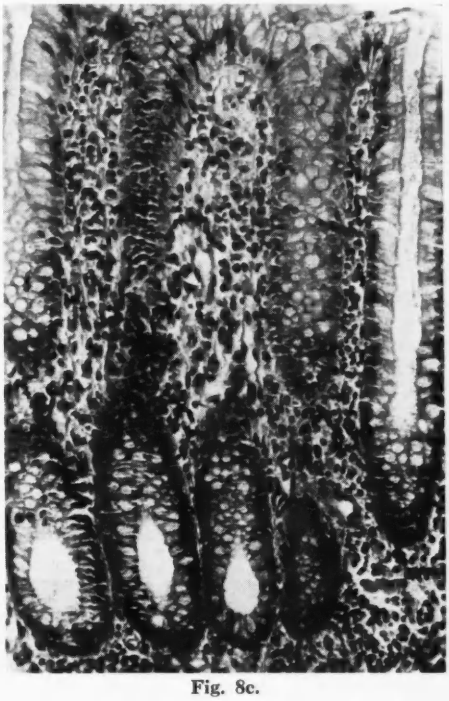


Fig. 8c.

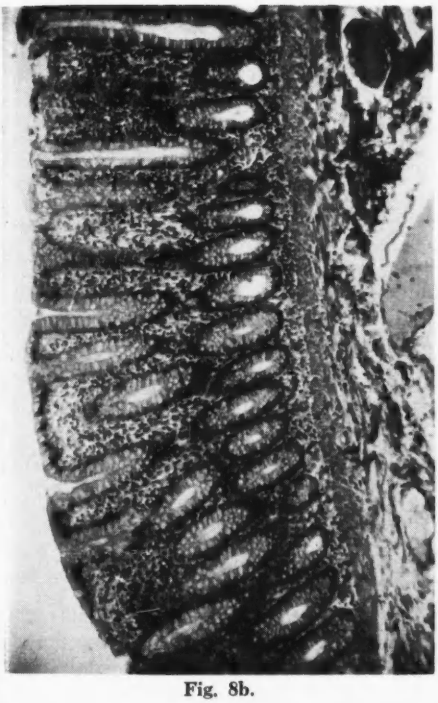


Fig. 8b.

Figs. 8a, b and c.—Biopsy of a neo-colic bladder made one year after operation: (**a**) Cross section of all layers, X 20; (**b**) Cross section of mucosa and submucosa, X 25.2; (**c**) Cross section of the mucosa showing mucosal cells, X 60.

anastomosis in those cases where the bladder resection has been done below the trigone. We dissect the ureters in the shortest possible length, so as to displace them in the least possible way from their normal situation, thus protecting their blood supply. The ureterocolic anastomosis is made at the tænia, a few centimeters from both extremities of the segment. Usually we utilize the Leadbetter technique because this procedure gives us the best results. *We never utilize indwelling ureteral catheters* for these ureterocolic anastomoses.

10. When all the anastomoses are finished, we verify that they are watertight by irrigating the newly-formed bladder through the urethral catheter.

11. With a continuous suture we then bring together on the median line around the mesentery of the newly-formed bladder the two flaps of peritoneum that had

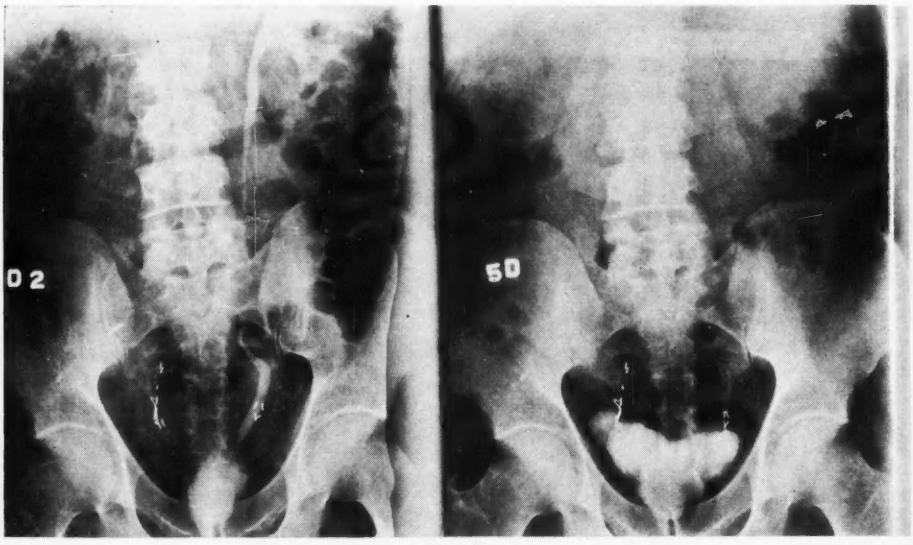


Fig. 9a.

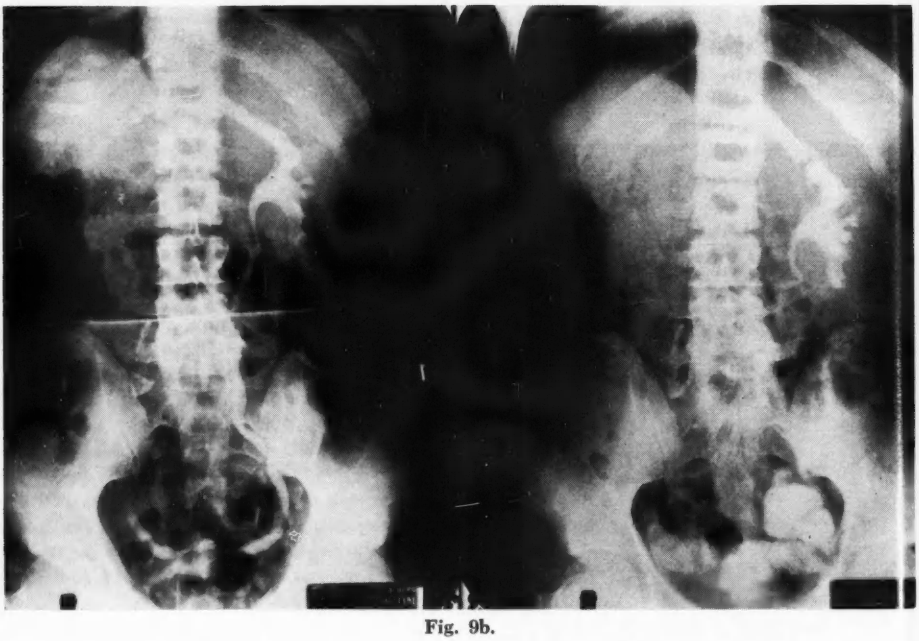


Fig. 9b.

Figs. 9a and b.—Results in two cases of colocystoplasty for small contracted bladders. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

been retained, so as to completely separate the retropubic space from the peritoneal cavity.

12. We then close the peritoneum after having instilled 2 g. of a streptomycin solution and drain the peritoneal cavity



Fig. 10a.

with a Penrose drain exteriorised through a paramedian counter incision so as to isolate the operative wound.

13. Two other counter incisions, also paramedian and suprapubic, left and right, bring out two Penrose drains placed in the retropubic space where from 2 g. to 4 g. of streptomycin in solution has been instilled.

14. We then close the abdominal wound in layers, after having placed from three to four reinforcing stitches including all the layers of the abdominal wall to avoid a postoperative hernia made possible by the length of the incision and the trauma produced by prolonged use of retractors.

15. An isolating dressing is placed on the wound and we apply changeable gauze dressings at the points of exit of the drains (Figs. 4, 5, 6, and 7).

Postoperative Care

1. We administer intravenously routine antibiotics and sustaining liquids.

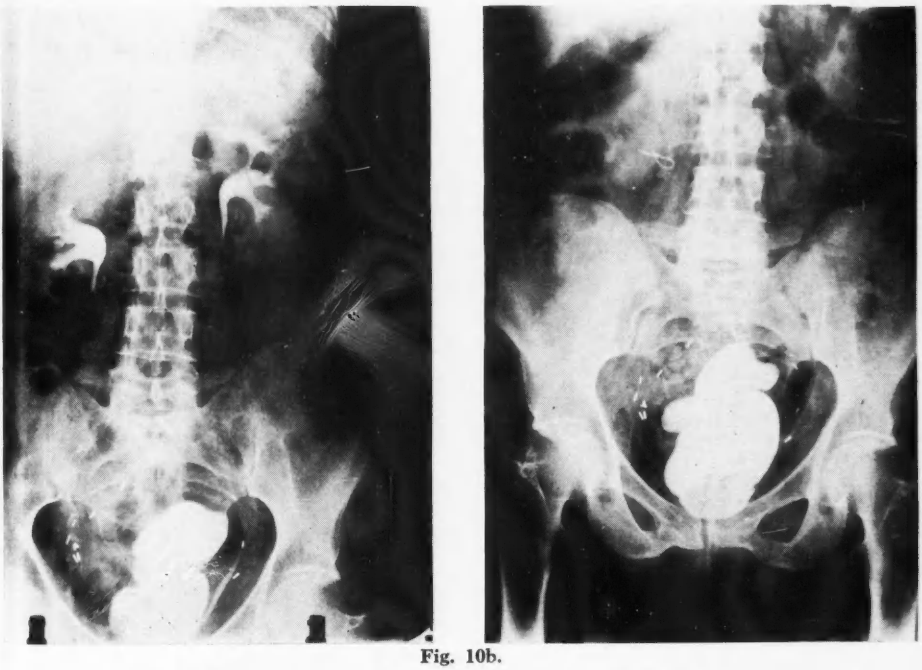


Fig. 10b.

Figs. 10a and b.—Results in two cases of colocystoplasty for interstitial and contractile cystitis: the last film on right is a forced retrograde cystogram showing no reflux. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).

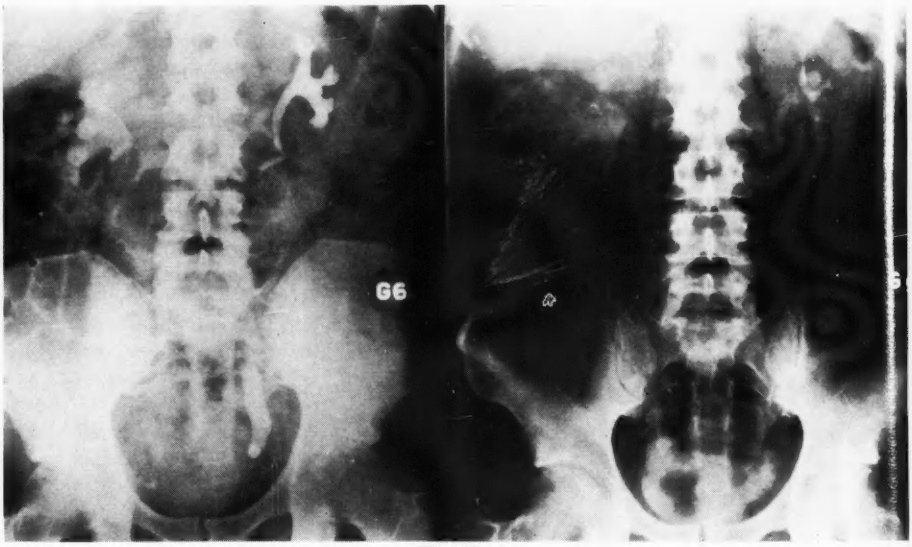


Fig. 11a.

2. We verify the proper functioning of the catheter by small irrigations made frequently under low pressure, mostly during the first 24 to 48 hours so as to be sure that no obstruction due to the mucus will strain the anastomoses during the period of consolidation. For such irrigations we use a 1% neomycin solution, to avoid infection as much as possible.

3. Continuous suction is installed on the

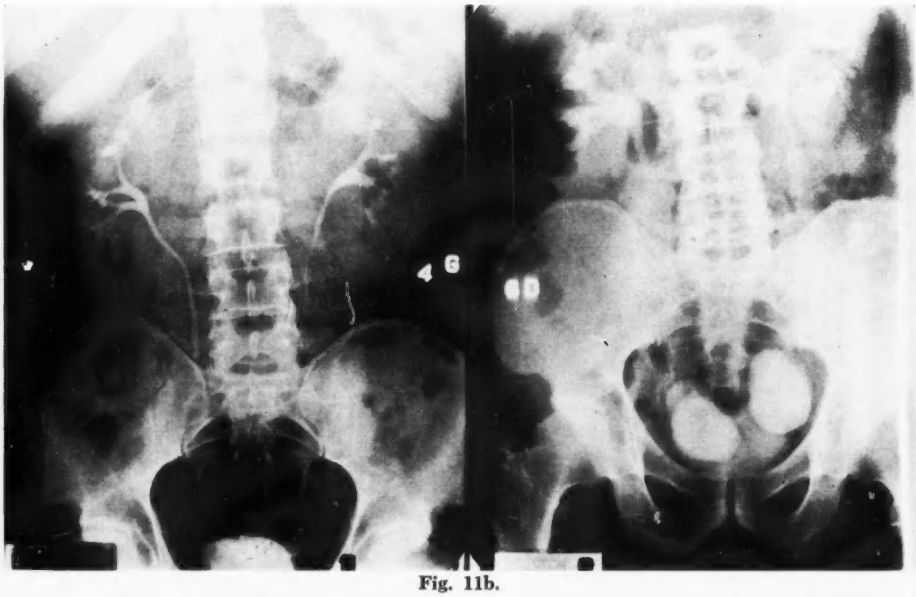


Fig. 11b.

Figs. 11a and b.—Results of colocystoplasty in two cases of cancer of the bladder one year after operation. (Reproduced by kind permission of the Journal of Urology, published by the Williams & Wilkins Company, Baltimore).



Fig. 12a.

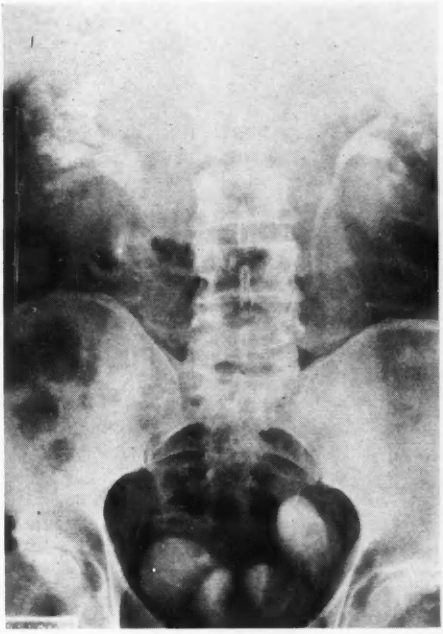


Fig. 12c.

Figs. 12a, b and c.—Preoperative film and results one and two years after colocystoplasty for cancer of the bladder.



Fig. 12b.

Cantor or the Levin tube according to the case.

4. Blood losses are replaced and electrolytes are controlled checking their levels every third or fourth day.

5. The Cantor or the Levin tube is removed on the third or the fourth postoperative day, as soon as the patient passes flatus. As soon as possible, we start to feed the patient with a non-residue diet. To all of our patients, to avoid postoperative stress ulcers or gastrointestinal irritation caused by the tube, we administer at intervals of three to four hours, an aluminum gel with mineral oil, to protect the gastrointestinal mucosa and facilitate the first bowel movement. This detail in postoperative management has always been a great help.

6. The patients are up on the third day, ensuring that the catheter remains open.

7. The peritoneal drain is shortened after 48 hours, and if in the next 24 hours no drainage is observed, it is removed. The retropubic drains are also withdrawn



Fig. 13a.



Fig. 13c.



Fig. 13b.



Fig. 13d.

Figs. 13a, b, c and d.—Results in four cases of colocystoplasty for cancer of the bladder three years after operation.

slightly after 48 hours, and thereafter are shortened by a few centimeters every day until they fall out spontaneously; this usually takes eight days.

8. The catheter is removed between the 12th and 14th postoperative day, to prevent fistula formation.

Results

Forty-five of these operations have been performed to date in the author's department; of this number 34 were done by the author, 10 by Dr. J. Charbonneau and one by Dr. G. E. Gauthier. We have operated upon 23 men and 22 women from 26 to 70 years of age for the following pathological conditions; (a) 14 cases of small contracted tuberculous bladders; (b) 15 cases of interstitial or retractile cystitis, and (c) 16 cases of bladder tumours where a total cystectomy was indicated; of these the substitution was made seven times to the beak of the prostatic capsule and nine times to the membranous urethra inside the external sphincter.

We have had up to now but one death, a mortality rate of about 2.3%; this was due to a cerebral accident on the seventh postoperative day, as shown by autopsy. All the other patients are alive and well.

Morbidity is practically non-existent. We have had (1) seven cases of fistulas which all healed spontaneously, the longest delay being 34 days and the shortest three days. One appeared 10 months after the operation; the sinus was resected and the recovery was uneventful; (2) five cases of electrolyte imbalance which was rapidly rectified and did not reappear; (3) all our patients have satisfactory micturition control and none is incontinent; in some cases there was a certain degree of nocturnal incontinence during the first few months, but it never persisted more than three months. In those cases in which the anastomosis was made to the urethra, there was a certain degree of urgency to which the patient adapted himself by getting to know the time of micturition intervals; (4) the patients urinate well, at reasonably regular intervals of three to four hours during the day, and once or sometimes twice during the night; (5) the mucus which is present in the first few months after the operation has a tendency to diminish and practically disappears after two years; (6) shown in Fig. 8 are microphotographs of a biopsy taken one year after operation on a patient operated on for cancer of the bladder. Very little change is evident in the mucosa. There is no metaplasia. A moderate degree of sclerosis of the submucosa, round cell infiltration of the lamina propria and a nearly normal muscularis are present. In fact, after one year there are only very minute changes in the different layers of the urine-containing segment (Fig. 8); (7) Figs. 9-13 show endovenous pyelocystograms of some of the different patients operated upon. All were made at least one year after operation.

Conclusions

Colocystoplasty for bladder enlargement or substitution is an operation that has been added to our therapeutic surgical arsenal; its indications are well established and its technical procedure is precise and proven. It is a serious operation and each case has to be studied and evaluated in detail before any decision to perform this procedure is made. It is a long, difficult and extremely meticulous operation, and although it is not the answer to all of the bladder problems, it is an operation which will render life worth living for many patients who would have died miserably before the advent of this fascinating operation which, although it has just been born, is already full of promise.

Résumé

La colocystoplastie est employée avantageusement pour agrandir ou remplacer la vessie dans: (1) La petite vessie tuberculeuse contractée, (2) les cas de cystite interstitielle rebelle avec ou sans ulcère de Hunner, (3) la cystite chronique sclérosante ou rétractile, (4) les cancers vésicaux opérables.

La technique opératoire est précise et a fait ses preuves. L'intervention est sérieuse, et chaque cas doit être étudié et évalué minutieusement. C'est une opération longue, difficile et extrêment méticuleuse, et quoiqu'elle ne soit pas la réponse à tous les problèmes de vessie, elle permet à plusieurs patients qui autrement n'auraient que le choix de mourir misérablement de poursuivre leurs jours.

CASE REPORTS

VENTRICULAR SEPTAL DEFECT DUE TO NON-PENETRATING CHEST TRAUMA: REPORT OF A CASE WITH SURGICAL CORRECTION*

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Introduction

A total of 21 cases of isolated ventricular septal defect due to closed chest trauma has been reported in the literature.[1] Two of these cases have been treated surgically with partial repair of the defects.[2, 3] This is the first reported case to have had complete correction of such a defect.

This case is presented to illustrate the diagnostic and symptomatic peculiarities of cardiac trauma and the relief of symptoms following repair of an isolated ventricular defect using cardiac bypass.

Case Report

A hard-rock miner aged 37 years, was in perfect health doing heavy duty, with no cardiac or other abnormalities recorded on previous physical examinations before his injury. On May 3, 1957, his chest was crushed between a three-ton bucket and a steam shovel Following four hours' loss of consciousness, the patient experienced severe chest pain and coughed up dark red blood. He was admitted to St. Joseph's Hospital, Blind River, Ontario, with a diagnosis of chest contusions and radiological evidence of a fractured left scapula.

No cardiac abnormality was suspected. He was treated with bed rest, oxygen, and sedation for two weeks. Following discharge the patient remained well until September 1, 1957, when, on rehabilitation activities, he experienced anginal type pain with dyspnœa which subsequently recurred on 75 yards rapid level walking, or climbing one flight of stairs.

In October 1957, examination demonstrated a grade 3 systolic murmur in the third left interspace radiating faintly to the neck, and an electrocardiogram suggested left ventricular hypertrophy. Aortic valvular disease was suspected at this time.

In May 1958, one year after injury, the electrocardiogram recordings were suggestive of mild myocardial injury. Treadmill exercise until development of chest pain, did not produce significant electrocardiographic changes.

On March 30, 1959, he had been off work for nearly two years and was experiencing an increase in his angina of effort. The murmur was somewhat louder and definitely pansystolic. This finding and a shunt pattern to the venous dye dilution curve led to consideration of a traumatic ventricular septal defect. Right heart catheterization confirmed the presence of a left to right shunt at ventricular level and the catheter was passed across the defect (Table I).

A Master's two-step test was normal. In view of the unusual presenting symptom of angina, a coronary arteriogram was performed to rule out coronary artery involvement. Good filling of both main coronary vessels was demonstrated.

On October 1, 1959, a 1 cm. septal defect was repaired through a right ventriculotomy using a bubble oxygenator and roller pump for total cardiac bypass. A heat exchanger incorporated in the circuit allowed cooling of the patient to 31° C. during a 21 minute period of perfusion. The defect, located in the anterior apical region of the septum, was repaired with interrupted silk sutures without the use of cardiac arrest. A median sterno-

TABLE I.—Preoperative Right Heart Catheterization, April 10, 1959

Site	Pressure, mm. Hg	Oxygen content Vol.%
Left pulmonary artery		12.15
Right pulmonary artery	23–24/6–10	12.15
Main pulmonary artery	26–31/6–9	12.75
Right ventricle, high	25–29/0	13.27
mid		12.39
low		12.51
Right atrium	6 (mean)	11.55
Superior vena cava		11.53
Inferior vena cava		11.53
Left ventricle	108–113/0	15.05

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tomy obviated the need for opening either pleural cavity.

The postoperative course was entirely normal and the patient was discharged three weeks after operation, on restricted activity.

He was readmitted for evaluation on February 10, 1960. At this time minimal aching pain related to the operative incision was the only complaint. There has been no recurrence of angina. A grade 1-2 systolic murmur persisted in the third and fourth left interspaces. An electrocardiogram demonstrated partial right bundle branch block. Venous dye dilution curves and right heart catheterization findings were normal (Table II). Treadmill exercise failed to elicit anginal pain or electrocardiographic changes other than a transient tachycardia.

A recent report, ten months after operation, indicated continued relief from symptoms.

Discussion

Cardiac lesions have been variously estimated as occurring in from 10% to 75% of severe cases of closed chest trauma.[4-6] Parmley[7] has applied the lower figure to automobile injuries occurring in the United States to obtain an annual incidence of 150,000 cases of cardiac trauma from this source alone. In his series of 546 cases of non-penetrating trauma to the heart examined at autopsy, Parmley reported five cases of isolated ventricular septal defects and 25 cases of septal rupture associated with other cardiac lesions.

The injury may be a direct force of a compressing or crushing nature applied to the chest wall, or due to violent displacement of the heart such as occurs in abrupt deceleration accidents. Cases are also reported following blast injuries.[7]

With direct injury to the chest the defect may occur immediately from compression of a distended chamber. Delayed development of a shunt is due to secondary rupture of a contused area of septum.

TABLE II.—Postoperative Right Heart Catheterization, February 17, 1960

Site	*Pressure, mm. Hg*	*Oxygen content Vol. %*
Left pulmonary artery		11.35
Right pulmonary artery	19–25/5–9	11.35
Main pulmonary artery	21–25/6–10	11.85
Right ventricle, high	24–26/0–2	11.35
mid		11.59
low		11.47
Right atrium	9 (mean)	11.59
Superior vena cava		10.66
Inferior vena cava		12.12
Femoral artery		16.29

The damage to the chest wall itself may be severe or, as in the patient described in this report, deceptively minimal. Diagnosis is made difficult by the associated chest trauma, shock, and delay in appearance of the classical cardiac signs. A daily clinical assessment of cardiac status is therefore mandatory for patients with any severe chest injury.

Anginal pain has not previously been reported in association with a traumatic ventricular septal defect, although the occurrence of angina pectoris following chest injury is not rare. It has been suggested that trauma can precipitate thrombosis in a diseased and arteriosclerotic coronary system.[8, 9] Parmley[7] and Moritz,[10] in extensive studies, failed to demonstrate the occurrence of coronary thrombosis following trauma to healthy vessels. They suggest that myocardial contusion, without occlusion of a major coronary vessel, may produce anginal type pain. An alternative explanation for the pain in our patient with a normal coronary tree is that, during exercise, increased shunting occurs across the defect with a consequent diminution in systemic and coronary blood flow.[11]

The prognosis in untreated traumatic ventricular septal rupture depends on the size of the defect. A large shunt may lead to intractable cardiac failure with rapid death. Urgent operation may be necessary, as in the case reported by Peirce.[2] Smaller defects will permit the gradual development of chronic right ventricular failure. A strong case can be made for repair of even the smaller, asymptomatic defects in view of the tendency for acquired arteriovenous communications to increase in size. One might expect an acquired defect to increase in size at a greater rate than a congenital defect with a more rapid progression of hæmodynamic changes.

In the case described, angina pectoris, without evidence of coronary artery disease or gross myocardial damage, was a presenting complaint. This has been completely relieved by closure of the defect.

The results of surgical treatment for traumatic injuries of the heart may prove very satisfying. In most instances traumatic septal defects will be of short duration with minimal pulmonary vascular changes which prove such a problem in the congenital variety of septal defects.

The surgical technique applied is that used for correction of the congenital lesion, with total heart-lung bypass using a bubble oxygenator and an occlusive pump. A heat exchanger in the extracorporeal circuit permits application of hypothermia during bypass with the benefits previously reported.[12]

The most common situation for traumatic ventricular septal defects is in the muscular septum, generally anteriorly at the apex. Defects may occur however, at higher levels and in the membranous portion. The defect approached from the right ventricle is more readily located in the trabeculated septum by maintaining cardiac activity. The danger of injuring conducting tracts during repair of these muscular septum defects is not great.

Summary

A case of traumatic rupture of the interventricular septum of the heart is described. This followed closed chest injury.

The presenting symptom was angina with progressive disability.

The septal defect was closed under direct vision with cardiac bypass. The patient has maintained complete relief of symptoms 10 months after operation.

A brief review of the literature is presented.

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Résumé

Nous avons décrit un cas de rupture interventriculaire à la suite d'un traumatisme thoracique. Il est à noter ici que la cage thoracique ne présentait aucune lésion communicative.

Le patient se plaignait surtout de douleur angineuse et d'incapacité croissante.

Nous avons réparé le septum par chirurgie à ciel ouvert au moyen de circulation extracorporelle. Le patient nous affirme maintenant qu'il est exempt de tout ennui depuis 10 mois et tous les tests de fonction pulmonaire et cardiaque se sont avérés normaux.

Notre description inclus aussi les differentes opinions concernant la conduite à suivre lors de traumatismes cardiaques.

SACCULAR ANEURYSM OF THE OPHTHALMIC ARTERY: REPORT OF AN UNUSUAL CASE

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WE HAVE BEEN UNABLE to trace previous reports of a saccular aneurysm arising from the ophthalmic artery. For this reason and because of the unusual clinical picture with associated trauma and monocular blindness this case is presented.

CASE REPORT

The patient was a 31-year-old Italian miner, brought unconscious from the depths of a mine in Thompson, Manitoba, following an explosion. The patient was an experienced mine worker and the blast was routine. There were no witnesses to his actions immediately before the explosion; hence it is not known whether he collapsed and was unable to get out of the way or just why he remained in the explosion area. He had a large contusion about the left orbit and multiple puncture wounds with imbedded foreign bodies about the face and hands but no evidence of trauma in the posterior part of his neck or head. Some three to four hours later he regained consciousness, and then rapidly sank into profound coma with a pulse rate in the low forties and an associated right hemiparesis. A trephine was performed in the left posterior parietal area which revealed fresh subarachnoid blood. Some 24 hours later the patient gradually began to rouse and remained conscious for the following seven days only to become deeply comatose again with an associated right-sided hemiplegia and a pulse rate in the low forties. The patient was then flown to the neurosurgical service at the Winnipeg General Hospital. On arrival he responded only to painful stimuli and could move only on the left side. Skull radiographs revealed a linear vertical fracture in the left frontal region as well as the posterior parietal trephine defect. An angiogram was done immediately on admission anticipating the presence of a large subdural hæmatoma on the left, but the vessels came out to the inner table of the bone and there was only minimal shift of the anterior cerebral artery from left to right. A saccular aneurysm approximately 3 mm. in diameter was visualized arising with a short stalk from the ophthalmic artery approximately 3 mm. beyond its origin from the internal carotid artery (Fig. 1a and b). The ophthalmic artery filled normally beyond the aneurysm stalk. Within the next 24 hours the patient gradually regained consciousness but was apractic and aphasic. Additional ophthalmologic examination revealed that the light reflex was exceedingly sluggish in both eyes presumably due to fine foreign bodies embedded within the cornea. It became doubtful whether there was any vision in the left eye. Fundoscopic examination revealed no abnormality. Within the next two days the man was sufficiently alert for accurate responses and it was apparent that his left eye was totally blind. A vertebral angiogram and a right carotid angiogram were both entirely normal.

Fourteen days following the initial accident, a left transfrontal craniotomy was performed. The aforementioned fracture line was visible at operation, extending down into the roof of the orbit where it crossed diagonally from the anterolateral aspect of the orbital roof across into the superior portion of the optic canal (Fig. 2). The optic nerve as it entered the canal was elevated sharply in passing over the proximal portion of the fundus of the aneurysm. There was no discolouration of the optic nerve but there was a firm thrombus protruding through the fundus of the aneurysm adherent to the overlying optic nerve. The adjacent brain was stained yellow. The optic foramen was uncapped and the adjacent orbital roof was removed for about 2 cm. The periorbital tissue was incised providing some mobility for the optic nerve. As the nerve was retracted medially the aneurysm burst. The ophthalmic artery was clipped proximal to the aneurysm stalk and additional clips were placed across the stalk after resecting the fundus. There was no interference with any of the collateral vessels and hence the operative procedure should have had no effect on the circulation to the nerve or to the retina. Inspection of the nerve revealed no evidence of any damage other than the local discolouration at the point of adherence of the above mentioned thrombus. There was no evidence of constriction or indentation of the nerve contour where it had rested on the aneurysm

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Fig. 1a.—Preoperative lateral views of angiogram. Upper arrow points down to fundus of aneurysm, lower arrow points up to continuation of ophthalmic artery into the orbit.



Fig. 1b.—Same view after operative removal of the aneurysm. The uppermost clips are on the dura.

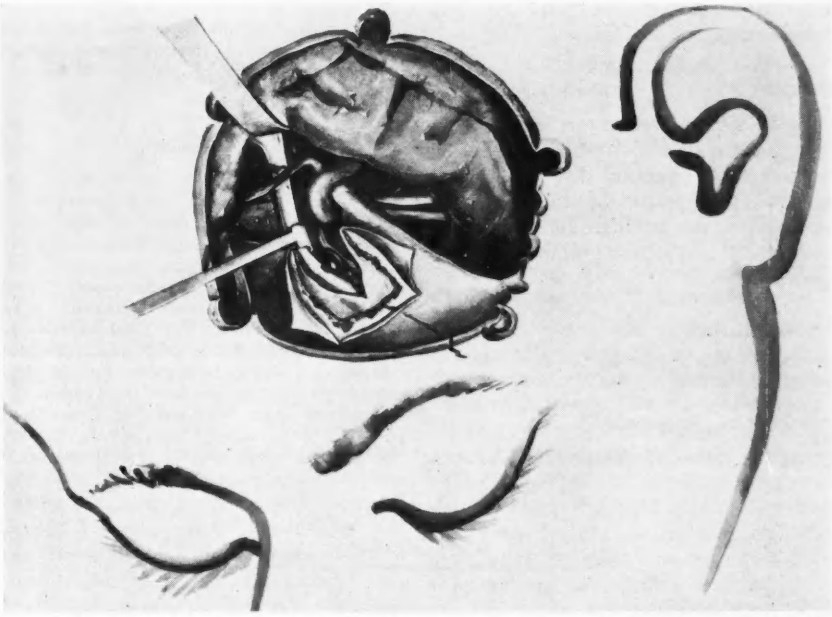


Fig. 2.—Artist's drawing from operative exposure. The proximal orbit is unroofed, the periorbita incised and the optic nerve retracted medially uncovering the saccular aneurysm which is seen to arise from a short stalk from the otherwise normal ophthalmic artery. The distal portion of the fracture line is portrayed, the proximal portion having been removed with the roof of the orbit. Note the thrombus protruding through the aneurysm fundus.

fundus, nor where it had passed through the optic canal in association with the aneurysm. The wound was closed and the patient made an uneventful recovery but has remained blind in the left eye.

Discussion

Aneurysms arising from the ophthalmic artery must be exceedingly rare. Aneurysms in this region with a stalk arising from the carotid artery are not uncommon and one such case was reported with an almost identical history of hæmorrhage and monocular blindness following trauma.[1]

The relationship of trauma to aneurysm rupture, particularly to delayed rupture, will always provoke some discussion. In the case of this miner the available information does not reveal whether spontaneous hæmorrhage caused the man to fall and hence to be in the path of the blast, or whether the blast rendered him unconscious and the subsequent fall fractured his skull which in turn started the aneurysm bleeding. The former sequence of events is suspected inasmuch as he was an experienced mine worker dealing with a scheduled routine explosion. It would seem that the man's subsequent course was due to his aneurysm. His second lapse into coma with a slow pulse and the third lapse 10 days later can be explained on no other basis, no subsequent subdural or extradural bleeding having been found.

The relationship of the trauma to the monocular blindness is also debatable. The fracture would most assuredly be credited with the monocular blindness were it not for the presence of the aneurysm, and the aneurysm in its turn would be credited were it not for the presence of the fracture. Obviously the quiescent aneurysm was causing no symptoms. The man gave no previous history of visual disturbance. Even after rupture the angiogram revealed no interference with the circulation through the ophthalmic artery and its branches, but the jet from the bursting aneurysm may well have permanently interrupted the function of the optic nerve in

addition to producing the other fluctuating neurologic signs.

SUMMARY

A case of saccular aneurysm arising from the ophthalmic artery, rupturing in close association with trauma, has been presented. The relationship of the trauma to rupture and of the trauma and rupture to the persistent monocular blindness were not proven.

ACKNOWLEDGMENT

The work of Miss Nancy Joy, medical artist, in preparing the drawings is gratefully acknowledged.

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RÉSUMÉ

L'anévrisme de l'artère ophthalmique est très rare. Le cas d'un mineur, âgé de 31 ans, sans signes oculaires antérieurs, est rapporté. A la suite d'une explosion minière le patient fut trouvé comateux, souffrant d'une fracture du crâne et de multiples contusions orbitaires. Une intervention chirurgicale révèla un anévrisme sacculaire émanant de l'artère ophthalmique gauche. Il y avait rupture du sac anévrismale, sans lésion proprement dite du nerf optique. Le malade est resté aveugle de l'oeil gauche. Il n'a pas été possible d'établir de lieu de causalité entre l'anévrisme et l'accident, entre l'accident et la rupture anévrismale, ou entre la rupture et la cécité monoculaire persistante.

DARWIN AND FREUD*

"Twice in a century, the human self-estimate has been rudely upset by the work of one man. A hundred years ago Charles Darwin showed us that we are not unique, and before the turn of the century Sigmund Freud had shown us that we have no insight into our own motives. As a result of this double blow to tradition, thousands of people all over the world now know, or think they know, that Darwin proved that men are descended from apes, and Freud that everything is really due to sex. The geographical and social range of those misconceptions is itself a measure of the size of the revolutions. . . .

"Both Darwin's and Freud's hypotheses were developed to explain empirical observations; both are in some particulars almost certainly wrong; both have been strikingly successful in organizing facts which were known but not understood when the theories appeared, and facts which have been discovered since. Both come within the ordinary scope of scientific method.

"After a hundred years, with a few powerfully motivated exceptions, most people now accept the main drift of Darwin's argument. After fifty years, if we take the essentials of Freud, infantile sexuality, the Œdipal reactions, dream-symbolism, defence mechanisms, and repression, some people will say, like Pasteur, 'Mon Dieu, sommes nous encore là?'; and others will angrily deny that any of them are verifiable at all. This is probably not very different from the situation of Darwinian anthropogenesis, perhaps not fifty, but certainly twenty years after the *Origin*. . . .

"Penetration of science in this way takes time. It has to wait for new evidence, for tempers to cool, and for ineducable experts to die. We know that Darwin until recently excited, and Freud still excites, the worst possible emotions in both opponents and proponents. We know also that psychoanalysis is a special case in that we are probably dealing with matter which is uniquely able to upset our judgment. This poses a problem in scientific argument which is new, quantitatively if not qualitatively, because we have to recognise the fact of so-called 'resistance', which we can easily verify in our own experience, and which makes it as hard for some people to consider the relevant ideas as it would be for them to see into their own ear. At the same time, we have to refrain from using this as an argument on theoretical questions, because it is obviously the joker to end all rational discussion".

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EXPERIMENTAL SURGERY

SURGICAL TECHNIQUE FOR HOMOLOGOUS TRANSPLANT OF NEONATAL KIDNEYS IN DOGS*†

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In recent years it has become appreciated that grafts of embryonic tissue do not elicit as great an antigenic response as adult donor tissue and are more likely to be accepted by a recipient.[1, 2] The most exciting of such reports deal with the successful transplants in humans, of fetal parathyroid tissue.[3, 4] The technical problems of transplantation in the adult have been overcome by numerous investigators and extensive studies have been made particularly with dogs, and more recently with sheep.[5-7] However, it seems that the omission of studies using fetal kidney transplants is significant. Indeed, in the Bibliography of Kidney Transplants recorded in the January 1958 issue of the *Journal of Plastic and Reconstructive Surgery*, there are 64 references from the world literature none of which cites experiments involving transplantations of fetal or neonatal kidneys.[8] The technique of anastomosis of renal vessels in the adult is difficult enough, but the small size of the renal vessels in the newborn precludes effective anastomosis. In dogs, the answer to this mechanical problem lies in the performance of the anastomosis at the level of the aorta and vena cava of the pup's donated kidneys.

It later came to our attention that the following attempts at neonatal transplants had been made. Mantelli, in 1913, transplanted the kidneys from pups to adult mongrel dogs using the aorta and vena cava of the pup anastomosed to the axillary vessels of the adult with early failure of all nine cases.[11] Dederer, in 1920, performed a renal homotransplantation between littermates, the recipient dying of distemper on the sixth day.[12] Murray *et al.*, in 1955, transplanted a kidney which had been ischæmic for two hours, into a 22-day-old pup. The kidney was still functioning at sacrifice 12 days later.[13]

A pilot study was undertaken in which the technical feasibility of this type of transplantation was evaluated and a series of 14 such transplants was performed. This study culminated in the evolution of the mechanically successful surgical technique to be described. There is sufficient blood supply from the periureteral branches of the renal artery to supply the trigone which can be joined to the adult bladder, thus solving the problem of drainage of the graft. In essence this type of graft entails the transplant of both kidneys, ureters and trigone of the bladder, and the anastomosis of the aorta and vena cava of the pup to the common iliac vessels of the adult host. The grafts have functioned well into the immediate postoperative period as confirmed by aortography, pyelography and urochromography. All dogs died or were sacrificed by the 17th postoperative day.

Surgical Technique

The technique to be described was evolved after 14 grafts, and it is considered that the following is a successful and practical technique for this type of transplant (Fig. 1). This entails the homologous transplantation of both kidneys, ureter and trigone with a 1 cm. segment of aorta and vena cava of a pup to the pelvic vessels of an adult dog.

Outline of Surgical Procedure

A. Donor (Pup)

1. Anæsthesia—Nembutal 10 mg./lb. by intraperitoneal injection.
2. Incision—pubis to xiphoid.
3. Eviscerate the pup.

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*First prize, Original Research, Scientific Exhibits, American Urological Association, Western Section, June 1960.

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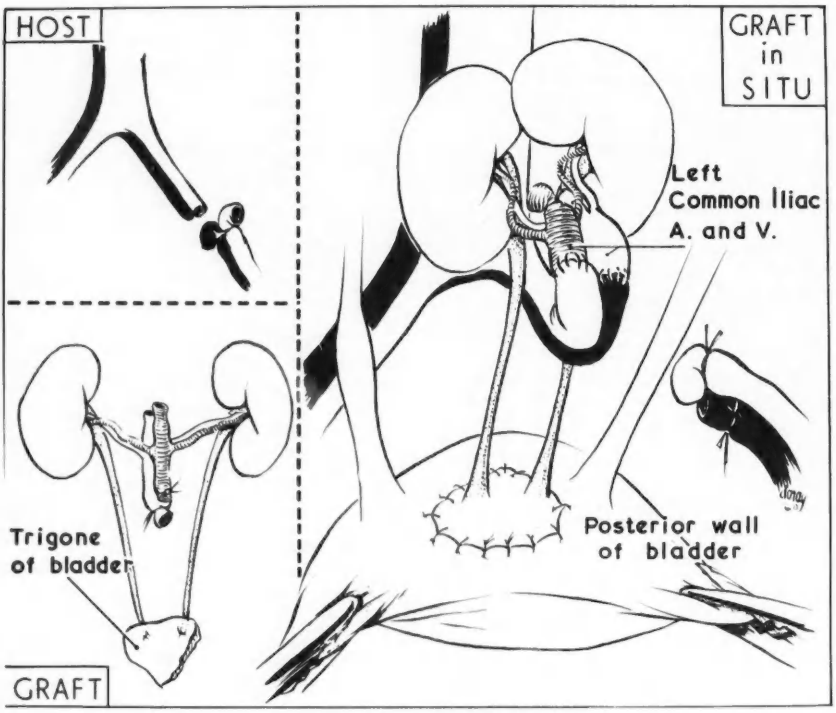


Fig. 1.—Surgical technique.

4. Mobilize the graft (bladder, ureters, kidneys).
5. Hypothermia.
6. Transect the aorta and vena cava.

B. Host

1. Anæsthesia — Intravenous Nembutal 10 mg./lb.—Pentothal (optional)—5% glucose in water by intravenous drip—intubate with oxygen.
2. Incision—left pararectus (dog)—midline (bitch).
3. Expose, mobilize, and divide the left common iliac artery and vein.

Detailed Technique

Two teams start simultaneously, one working to expose the left common iliac artery and vein of the dog host through a left pararectus (midline in the female) transperitoneal approach, and the other to obtain the neonatal graft. For anæsthesia, intravenous Nembutal 10 mg./lb. body weight is instilled slowly and a continuous intravenous drip of 5% dextrose and water is started. Intermittent positive pressure oxygen is administered through an endotracheal tube. As necessary, but with caution, increments of Nembutal, 25 mg. to 50 mg., are given intravenously to maintain proper anæsthetic levels. The posterior parietal peritoneum lateral to the rectosigmoid is incised, and the left common iliac artery and vein are identified and mobilized for at least 3 cm. to 5 cm. distal to the bifurcation. Using a right-angle clamp distally, as near to the inguinal ligament as possible, and a rubber-shod bulldog clamp proximally, the vessels are divided and separately ligated with 0 chromic catgut. The proximal ends are cleared of perivascular adventitia leaving a clean vessel wall for the anastomosis. When hæmostasis is satisfactory and the vessels thus identified, mobilized, transected and cleared of adventitia, the recipient site is considered ready.

The neonatal donor is anæsthetized with

Nembutal 10 mg./lb. body weight given intraperitoneally and the abdomen is entered through a pubis to xiphoid incision. Since the proposed graft is retroperitoneal, evisceration of the pup is performed by dividing the rectum, mobilizing the rectum and colon and dividing their blood supply. The small gut and stomach are similarly mobilized and their vessels ligated; the œsophagus is clamped, ligated and divided below the diaphragm. The bladder, ureters and kidneys are mobilized from below upwards until the kidneys are attached only by their pedicles. After injecting the vena cava with heparin 0.5 c.c., the aorta and vena cava are ligated and transected below the renal pedicles.

By maintaining slight traction on the kidneys, ureters and bladder, the aorta and vena cava are mobilized for a short distance superiorly, great care being taken to clamp and tie the lumbar vessels individually. The graft is then considered ready and the abdomen is packed with sterile chipped ice for 10 minutes. This local hypothermia tends to prolong the safe avascular period necessary for anastomosis. The aorta and vena cava are now divided proximal to the renal vessels and the graft is transferred to the pelvis of the host.

The vascular anastomosis is performed with 000000 atraumatic silk using a continuous everting Carrel suture. The thin friable veins are best joined by placing three sutures to form a triangle and closing between these stay sutures. It is necessary to join the sturdier arteries first, as this anastomosis splints the friable venous junction. When both anastomoses are complete, the bulldog rubber-shod clamp on the common iliac vein is first released; the back flow enables visualization and closure of any leaks. The artery clamp is then removed and the small aortic branches which may have been missed are now tied. Within two to three minutes of reconstitution of the vascularity to the graft, the ureters begin to fill and peristalsis is seen. Indigo carmine usually appears within six minutes.

Attention is then directed to the pup's bladder which is trimmed to within 1 cm. of the trigone. The posterior bladder wall



Fig. 2.—Dog U-14 sacrificed at 17 days. Adult bladder showing "take" of trigonal graft in upper right-hand corner (confirmed by microscopic section). Note ureters distended with urine.

of the host is cleared and a small button of full-thickness wall is cut out. Continuous 0000 chromic atraumatic suture is used to join the bladders. To place the recipient bladder at rest a No. 16 red rubber catheter is brought out anteriorly in the midline and cut flush with the skin as a cystotomy. This is attached to skin by 00 silk sutures. The ureters and vessels are placed loosely in the host's pelvis and covered over with sigmoid mesocolon and omentum. We feel that torsion of ureters and/or vessels is thus less likely than if nephropexy and reperitonealization are performed. Closure of the abdominal wall is performed in the routine fashion after the host's left kidney is removed.

TABLE I.—Donor Statistics

Age	12-35 days
Weight	1½-5 lb.
Aorta diameter	3-4 mm.
Vena cava diameter	4-7 mm.

The dog is permitted to be ambulant and is given a fluid diet on the first postoperative day and a regular diet thereafter. Penicillin, 300,000 units daily, is given for the first five days postoperatively.

Observations

Our best time for anastomosis of the graft, and hence the period of avascularity of the graft, has been 15 minutes (five minutes for arterial and 10 minutes for venous anastomosis). In all, two hours seems sufficient for the total surgical procedure.

Pathologic study of the grafts taken after either death or sacrifice of four of the animals revealed the following:

Dog U-8. Bilateral complete hæmorrhagic necrosis and infarction. Dog sacrificed eight days postoperatively. There was a large clot in the arterial anastomosis.

Dog U-11. Bilateral calcinosis of nephrons and nephrosis. Bilateral hæmorrhagic necrosis and sloughing of the medulla. This dog died nine days after the graft. Yellow urine was seen in the renal pelvis which demonstrated a moderate degree of hydronephrosis.

Dog U-12. Pup's kidneys showed chronic interstitial nephritis. The host's kidneys were completely normal, showing no effect from the graft. This dog eviscerated on the sixth postoperative day and was sacrificed.

Dog U-14. The kidneys of this pup showed acute interstitial nephritis and acute hæmorrhagic cortical necrosis. The trigonal graft had taken well and was well vascularized. Both ureters appeared patent and there was a non-specific chronic cystitis and ureteritis noted in the graft. Vascularity was excellent. This animal was sacrificed on the 17th postoperative day (Fig. 2).

Radiography. An aortogram was performed on one dog immediately after operation and outlined clearly the vascularity to the graft. Twenty minutes after the aortogram, an intravenous pyelogram revealed excretion of the dye by the kidney, indicating that the anastomosis was functioning in the immediate postoperative period (Figs. 3, 4 and 5).

Since the purpose of this pilot study was to devise a surgical technique for this type of anastomosis, no attempts were made to study renal function and urinary output. The systemic effects of the fetal homo-

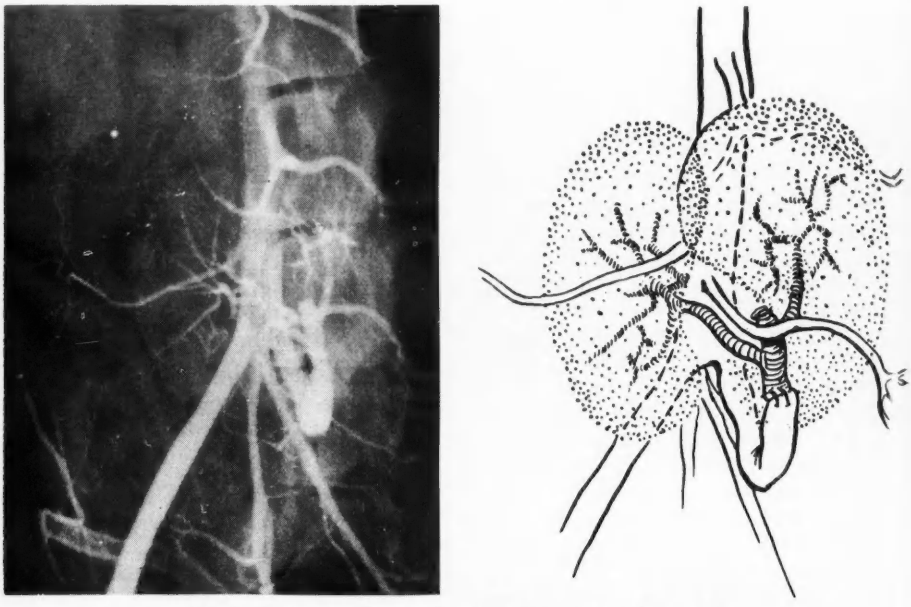


Fig. 3.—Aortogram showing functional vascularity of the graft.

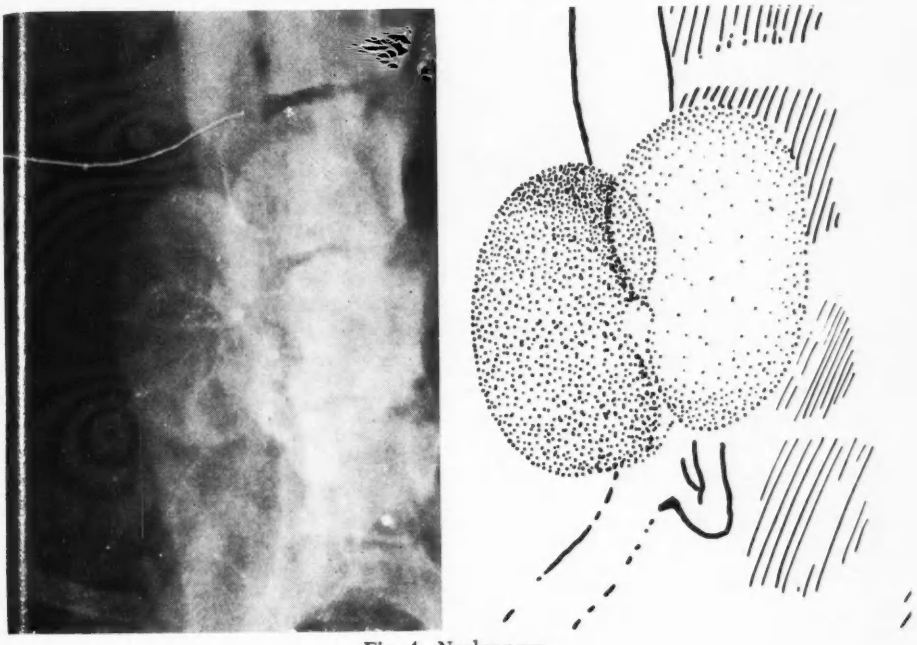


Fig. 4.—Nephrogram.

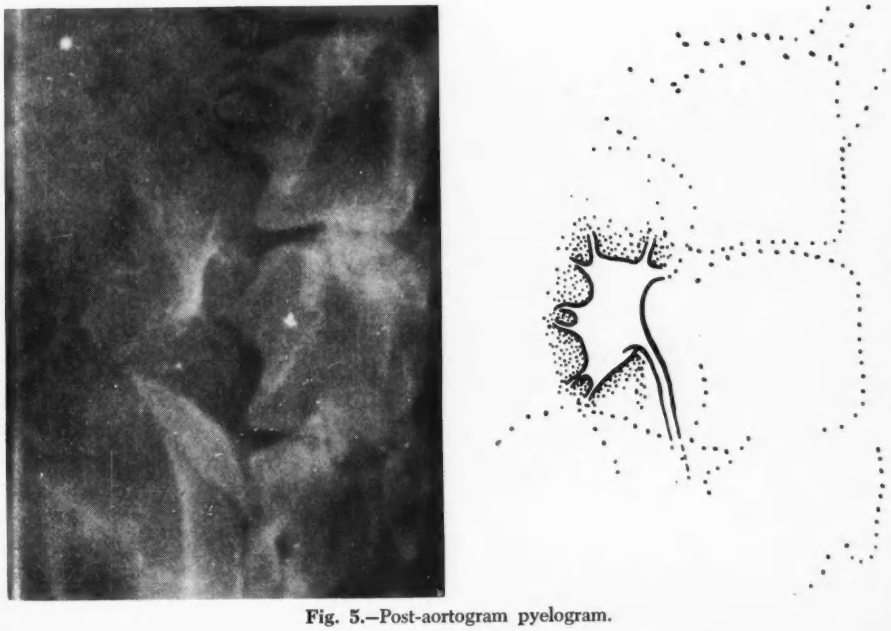


Fig. 5.—Post-aortogram pyelogram.

grafts appeared to be minimal compared to the anurias and toxic states described following adult homografts.[9, 10]

Summary

The answer to the mechanical problem of neonatal renal transplants lies in the performance of the anastomosis at the aorta–vena-cava level of the pup's donated kidneys. The technical feasibility of this type of transplant was demonstrated in the preliminary series of 14 transplants from pups to dogs. There is sufficient blood supply via the periureteral branches of the renal artery to supply the trigone, as proven by trigonal graft take. This new surgical technique warrants further evaluation. Additional studies are under way, with the hope that this approach may provide the springboard from which may come some contribution to the solution of the renal transplant problem.

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Résumé

On sait que les greffes de tissu embryonnaire occasionnent moins de réactions antigéniques que les greffes de tissu adulte. La transplantation rénale chez les animaux nouveau-nés n'a pas été bien étudiée, principalement à cause des difficultés techniques les vaisseaux rénaux se prêtant mal à la suture.

Les auteurs, dans une série préliminaire de quatorze chiots, ont pu perfectionner une technique permettant la transplantation homologue rénale dans un temps opératoire d'environ deux heures. La transplantation de l'unité reins-uretères-et-trigone de la vessie, enlevés en bloc avec un segment aorte-veine cave en amont des vaisseaux rénaux est prélevée sous conditions chirurgicales et greffée chez le chien adulte.

La technique est décrite en détail. Ce travail démontre les possibilités pratiques du procédé. L'étude de l'état des reins est laissée à un travail ultérieur.

IN DEFENSE OF TELEOLOGY*

"Teleology serves, . . . primarily as a guide to research. Whether conscious or unconscious, it sets up signposts in the blind wilderness of Facts. 'It can give direction to experimental design.' The researcher is not merely interested in the effect of *A* on *B per se;* he believes (or hopes) that *A* causes a such-and-such effect on *B* because it would fit in with his preconceived notion (hunch) that some causal relationship exists between these two. The dissatisfaction in finding no statistical or other relationship between *A* and *B* often leads to abandonment of the study—and to publication of the negative result. The very fact that negative results are reported much less frequently than they occur in the laboratory or clinic is an indication that the teleologic urge was not appeased by the data . .

"Let us recognize teleology for what it is – an intriguing, stimulating beacon in the darkness of our ignorance . . ."

*Editorial.: *Perspectives Biol. Med.*, **4:** 1, 1960.

THE HEALING OF UNTREATED EXPERIMENTAL PENETRATING GASTRIC ULCER*

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Dimpster[2] has suggested that the pathogenesis of human peptic ulcer resolves itself into three main problems: (1) Factors producing the ulceration, (2) factors accounting for localization of the ulcer, and (3) factors responsible for the delayed healing of the ulcer. Most of the studies of experimentally produced ulcers so far reported[1, 3-6, 8-12] have concerned themselves with superficial ulcers and thus deal primarily with mucosal regeneration. The method of producing a standardized penetrating gastric ulcer in the rat by thermocautery has been previously reported by Skoryna, Webster and Kahn.[13] The resulting defect involves the full thickness of the stomach wall and, during the healing phase, bears a close histological similarity to human peptic ulcer. It lends itself well to a study of the healing phase of penetrating gastric ulcer and the effects of physiological and pharmacological factors on this process.

In order to assess properly the effect of these factors on the repair process, a detailed chronological study of the healing of the experimental ulcer in untreated animals was undertaken on a large series of animals. The sequence of events following the production of the ulcer, including the inflammatory response, fibroplasia and the mucosal regeneration, was studied at intervals in rats followed up for 100 days after the production of the defect. The present report deals with the detailed histological sequence of repair in this type of lesion. It includes a comment on spontaneous reulceration and chronicity, and on their possible significance in relation to observations on human ulcers.

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This work was supported by a grant-in-aid No. MBT755 from the National Research Council of Canada.

Materials and Methods

A total of 300 male rats of the Royal Victoria Hospital strain, weighing 180 g. to 200 g. at the time of operation were used in this experiment. They were maintained on Purina meal diet and water *ad libitum*. Ulcerations measuring approximately 6 mm. in diameter, were made in the mid-portion of the posterior wall of the glandular stomach proximal to the pylorus, according to a method previously reported.[13]

The animals were sacrificed in groups at six, 12, 24, 48 and 72 hours, then at three-day intervals for the first month and then every 10 days up to 100 days. The specimens were fixed in formalin and stained with hæmatoxylin-eosin, Masson's trichrome, and periodic acid-Schiff techniques.

Results

Gross.—At autopsy, the ulcers in the first few days were slightly depressed and corresponded in size to the cauterized area. There was an inflammatory reaction over the serosa in the region of the ulcer. From this time on, in most instances, this region was in contact with, and often adherent to, liver, pancreas, omentum, spleen or other neighbouring structures. By the sixth day the ulcers were deep and sharply defined, with raised edges, had necrotic appearing debris on the surface, and thickening of the wall in the area of ulceration. By the 12th day the defect was still evident and the region of the ulceration was still thicker than the adjacent gastric wall. At 21 days, two-thirds of the ulcers appeared completely healed grossly; the area was still thicker than the adjacent stomach wall. At the end of one month, all the ulcers were healed on gross examination. From this time the site of ulceration became more and more difficult to locate but usually could be recognized by an area of scarring and puckering of the wall, by serosal adherence to a contiguous organ,

and by the stellate arrangement of the mucosal folds.

Microscopic.—The findings are summarized in Table I.

Six hours.—The stomach wall is intact, with the burned area depressed slightly from the surrounding unburned wall. The burned area consists of a wide zone of coagulative necrosis which involves the mucosa, submucosa, muscle coats and in many instances reaches the serosa. Here the general architecture of the gastric wall is distorted, shrunken, and most of the cytological details are lost.

Reactive changes are seen in the unburned tissue at the margin; there is marked hyperæmia and œdema of the submucosa and subserosa, with some fibrin strands and a few leukocytes. The surrounding surface mucosa shows increased mucus secretion. The muscularis mucosae ends abruptly at the margin of the ulcer. In the muscle coats, at the edge of the lesion, the muscle fibres stain brightly eosinophilic and form a syncytial mass in which the nuclei are hyperchromatic.

Twelve hours.—The defect consists of a wide zone of coagulative necrosis occupying the full thickness of the wall. At the edges the intact mucosa is raised by submucosal œdema and is bent downwards and leans towards the defect. The subserosal œdema has also increased. Neutrophils are more numerous and some extend along the serosal surface. Some of the vessels at the margin of the defect have damaged walls and are thrombosed.

Twenty-four hours.—(Figs. 1 and 2). The overall picture resembles that seen at 12 hours. The inflammatory response in the adjacent wall is now well marked; there is early penetration of leukocytes into the necrotic tissue and a fibrinopurulent exudate extends over the serosal surface. Young fibroblasts are present in the subserosa and submucosa of the bordering zone.

Forty-eight hours.—There is a definite area of ulceration involving all coats with the floor of the ulcer at the level of the serosa. The inflammatory infiltrate is marked. In the surrounding undamaged mucosa the glands are densely crowded and show prominent mitotic activity with as many as 10 mitoses per high power field; some of the deeper glands are dilated. From the undamaged mucosa a single layer of flattened cells extends over the ulcerated area for a short distance. Some of these cells have mucus in their cyto-

TABLE I.—The Histological Findings Seen at the Different Time Intervals in the Study of the Healing of Untreated Experimental Penetrating Gastric Ulcer

	Œdema	*Neutrophils*	*Necrotic material*	*Granulation tissue proliferation*	*Collagen*	*Mitoses in epithelium*	*Epithelialization of defect*	*Glandular differentiation*	*Contraction of site*	*Parietal cells*
6 hours	+++	+	++++							
12 "	++++	++	++++							
24 "	++++	+++	++++			⊥				
48 "	++++	+++	++++	+		⊥⊥⊥⊥	⊥			
3 days	++++	++++	++++	+		++⊥⊥	+			
6 "	++	++++	++	++++	+	++++	++			
12 "	+	++	+	++++	+	++++	++			
15 "		+	+	++++	++	++++	++	+		
21 "			+	+	+++	++++	+++	++	+	
28 "					++++	++	++++	++	++	
40 "					++++	+	++++	+++	++	
50 "					++++		++++	+++	++	
60 "					++++		++++*	+++	+++	
80 "					++++		++++	++++	++++	+
100 "					++++		++++	++++	++++	++

Legend: *Superficial mucosal re-ulcerations were found from this time on.
+ to ++++ grading of intensity of findings.

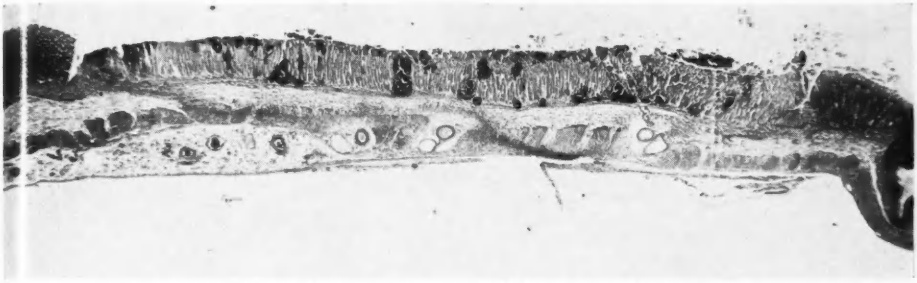


Fig. 1.—General view of coagulated area at 24 hours. There is full thickness coagulation necrosis of the stomach wall and early inflammatory exudation (H & E x 35).

p asm suggesting that they are differentiated cells from the surface which have sl d or have been pushed over from the adjacent margin. This single layer of epithelium lies on a bed of necrotic, granular, eosinophilic tissue which is infiltrated by neutrophils. One of the neighbouring viscera is usually found in contact with the serosal aspect of the burned area and from this time on, forms part of the base of the ulcer.

Day 3.—In the ulcer site the number of neutrophils in the necrotic tissue has increased; the latter shows dissolution into irregular eosinophilic fragments. In the marginal submucosa and subserosa a short distance behind the advancing leukocytes, there is marked fibroblastic activity and an interlacing network of cells is formed. A very occasional mitosis is seen in the viable muscle coat a short distance from the margin.

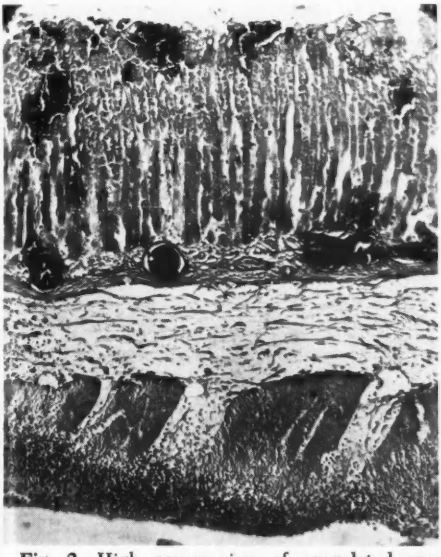


Fig. 2.—High power view of coagulated area shown in Fig. 1. The acellularity of all layers and the exudate on the serosal surface are evident (H & E x 90).

Day 6.—The surface of the ulcer at this stage is still depressed. The floor of the ulcer consists of a thin zone of fibrinopurulent material covered by a layer of residual necrotic tissue. The wall in the area of ulceration is thickened up to two to three times normal (Fig. 3). The increased thickness has resulted from a marked proliferation of granulation tissue at the base. Within the granulation tissue large capillaries course perpendicularly upwards from the serosa towards the surface where they become smaller and lose their orientation. A small number of collagen fibres are present in the deepest part of the ulcer wall and at the edges. The row of epithelial cells extending over the ulcer is longer and the glands at the margin still show signs of active proliferation (Figs. 4 and 5). Most of the mitoses are in the deeper parts of the glands. The submucosal œdema appears to be decreased.

Day 12.—The ulcer shows a well advanced healing phase both as regards connective tissue replacement of the necrotic wall and mucosal regeneration (Fig. 6). The central part of the regenerating epithelium consists of a single layer of flattened cells which merges peripherally with a layer of columnar cells; these have oval nuclei, prominent nucleoli, deeply basophilic cytoplasm, and occasional mitoses. Near the ulcer edge the columnar layer becomes stratified and between this and

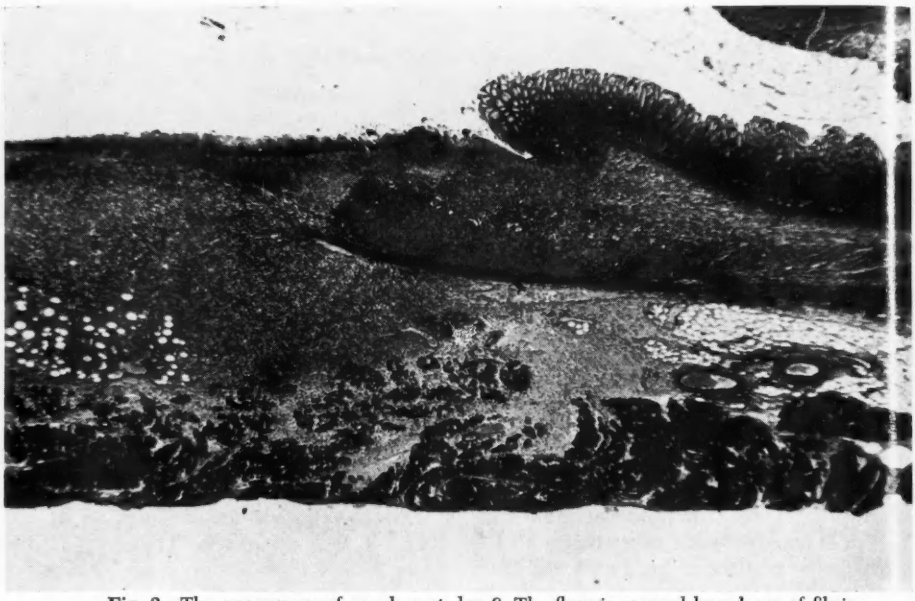


Fig. 3.—The appearance of an ulcer at day 6. The floor is covered by a layer of fibrinopurulent material. The transition between normal gastric wall and the ulcerated area is well demonstrated. The ulcer area is thickened as a result of a considerable amount of young granulation tissue. Pancreatic tissue is adherent to the serosal surface (H & E x 25).

the undamaged mucosa, the newly formed epithelium is invaginated into pits or foveolae which are small and shallow but get larger and deeper as the normal mucosa is reached. Mitoses are numerous in this area. The undamaged mucosa at the edge has deep pits composed of mucus secreting cells. The muscularis mucosae stops abruptly at the edge of the ulcer. The muscle coat also ends close to the edge but is pulled up so that its upper fibres join the muscularis mucosae. This completely seals off the uninvolved submucosa and also seems to pull the uninvolved marginal mucosa and serosa into the area of the defect, decreasing its extent in these two areas.

Day 15.—Granulation tissue proliferation is still prominent, but mitoses are fewer. In the areas in which fibroplasia first appeared, there is a decrease in cellularity and collagen fibres are most numerous and thickest.

Day 21.—The ulcer area remains thickened. Those ulcers which measure 6 mm. or less in width are completely epithelialized (Fig. 7). Those which are larger also show approximately 6 mm. of epithelial coverage. The epithelium covering the ulcer area has increased in complexity of structure. Close to the edge there are deep foveolae with communicating simple glandular structures; the surface cells and those of the foveolae are tall columnar cells with goblet-like vacuoles above their nuclei. The lamina propria of this mucosa has a rather loose stroma which contains small round cells. As the muscularis mucosa has not regenerated, this new mucosa rests on a granulation tissue base.

Day 28.—The ulcers are completely covered by epithelium and the gastric wall in the region has contracted to a degree and forms a lentiform swelling. The regenerated epithelium shows further differentiation so that deep to the foveolae are small acini or dilated tubules lined by low columnar cells with oval nuclei and pale granular cytoplasm. A few of the glands are cystic. The overall appearance of the regenerated mucosa is similar to that normally seen in the pyloric part of the stomach (Fig. 8). Beneath it, the wall

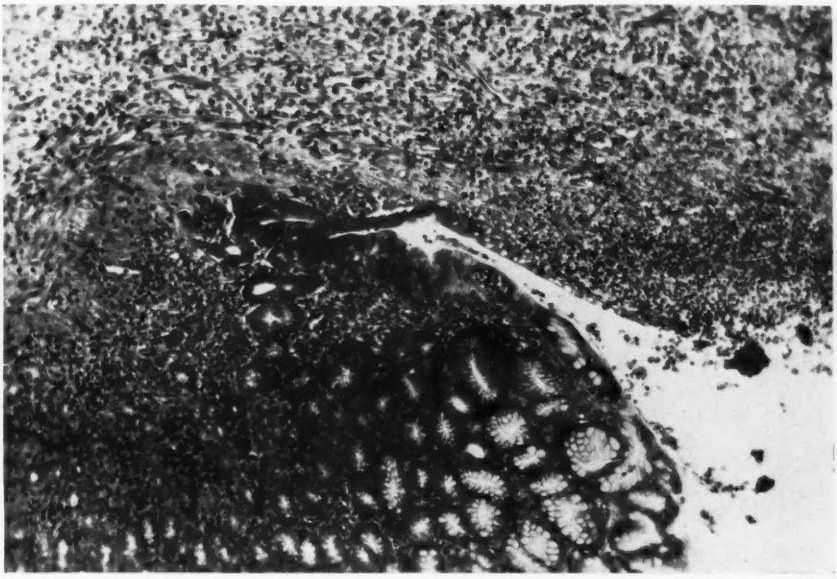


Fig. 4.—High power view of the edge of an ulcer at day 6. There is a row of epithelial cells extending over the edge of the ulcer floor. Some of these cells contain mucus suggesting that they are differentiated cells which have slid over from the adjacent mucosal surface. There is marked mitotic activity in the deep part of the normal mucosa at the ulcer edge (H & E x 100).

consists of a band of granulation tissue merging into a fibrous tissue base in which the collagen bundles lie mostly in a horizontal plane.

Day 40.—All the ulcers are well healed and less conspicuous (Fig. 9). There has been a further increase in the amount of collagen in the wall; tongues of dense fibrous tissue extend into the lamina propria. The ulcer site has contracted with reduction both in the width of the ulcer and in the thickness of the wall.

Day 50.—The stomach in the ulcer area consists of a dense fibrous tissue wall covered by a layer of intact mucosa. The latter has increased in thickness and in the main still resembles that of the pylorus; in places, however, glandular proliferation has continued and aggregates of glands extend deeper into the connective tissue wall in a manner reminiscent of Brunner's glands in the duodenum. In the muscle coats close to the margins of the ulcer, numerous mast cells are found. *This is the first occasion on which any number of these cells were found.*

Day 60.—A number of the healed ulcers have microscopic areas of surface necrosis (Fig. 10) located at or close to the centre of the regenerated mucosa. These vary from a small superficial mucosal erosion to a small focal ulceration penetrating a short way into the superficial portion of the connective tissue wall. The surface of these mucosal erosions is covered by a fibrinous exudate and they are surrounded by mild inflammatory reaction. That these are areas of superficial acute re-ulceration is shown by the reappearance of an inflammatory exudate and by the thickness and pattern of the differentiated epithelium surrounding them.

Days 80-100.—The ulcer site has become relatively inconspicuous but the defect in the muscle coat and the muscularis mucosae, the dense fibrous wall and the type of mucosa serve to differentiate the injured area from the normal. Some areas of superficial erosion are still found. The regenerated mucosa now resembles very closely the normal fundic mucosa of the stomach. The gastric glands have become orientated,

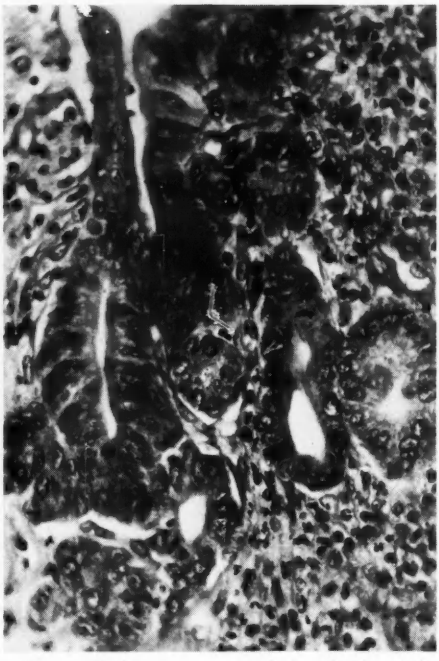


Fig. 5.—High power view of the glands of the mucosa at the edge of an ulcer at day 6. Numerous mitotic figures can be seen in the epithelial cells (H & E x 350).

and eosinophilic staining cells resembling parietal cells are present in some of the glands (Fig. 11). The ulcer site is now of the same thickness as the normal stomach, indicating that further contraction has occurred, and the measured width of the defect is 50% of its original size.

Discussion

The healing phase of the described experimental ulcers histologically resembles in many aspects that of healing peptic ulcers; both are deep ulcers penetrating through the full thickness of the stomach wall. They soon acquired a punched-out appearance with steep walls, little or no undermining, and a red base covered by necrotic and fibrinopurulent material. As healing proceeded there was fibrous replacement of the remains of the muscle coat and regeneration of the mucosa. The muscle coats including the muscularis mucosae failed to regenerate, so that the regenerated mucosa rested at first on inflamed granulation tissue and eventually on dense scar tissue.

The healing process observed was a well co-ordinated sequence of events which reproduced itself with striking uniformity throughout the whole series. With an ulcer of standard size it was possible to judge the age of the ulcer with great accuracy from its histological characteristics (Table I). As in wound healing elsewhere, there was an initial inflammatory reaction and a lag period as far as fibroblastic proliferation was concerned. This was followed by a surge of proliferation of granulation tissue which rapidly filled the defect and later bulged above the level of the surface of the surrounding normal mucosa. This was gradually and progressively converted into dense, poorly vascularized scar tissue. Concomitant with the repair process in the wall, mucosal regeneration was taking place. The first indication of the latter was seen at the edges of the ulcer wall after 24 hours. It followed the pattern described by other investigators.[4, 5, 10, 13, 14] Epithelial proliferation was manifested by mitoses and a crowding of the cells in the glands; some of the surface cells became flattened and rearranged, so that they extended over the periphery of the defect as a single flattened layer. As extension continued over the defect the more proximal parts of the regenerated epithelium showed invaginations with the formation of pits, which with further growth differentiated into a superficial mucous surface layer, with simple tubular glands. The process continued until the defect was completely covered. Progressive differentiation occurred in the older areas of the regenerated mucosa. The glands at first resembled pyloric glands, but with continued proliferation they resembled Brunner's glands. By 80 days, parietal cells and chief cells were apparent, and became more numerous as further differentiation occurred. Other investigators have similarly observed parietal cell differentiation in animals followed up for a sufficiently long period of time.[11] The regenerated mucosa resembled closely that of the normal fundic mucosa, except that this new mucosa lay directly on scar tissue without the interposition of a muscularis

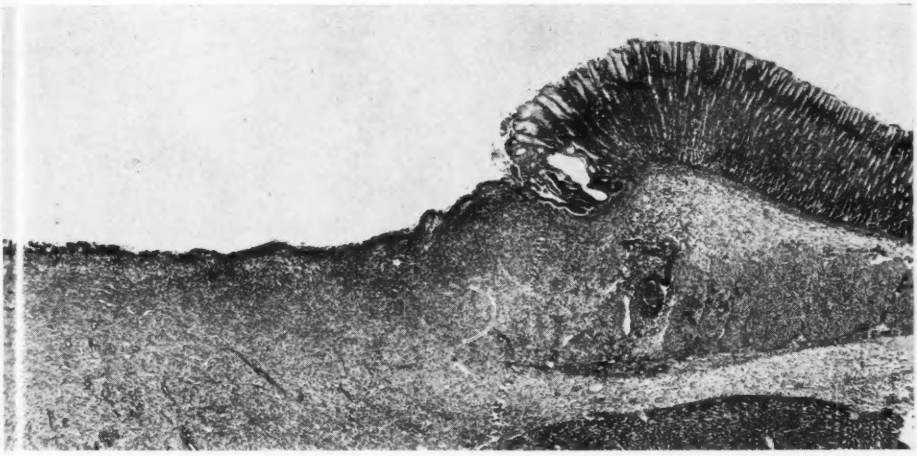


Fig. 6.—Low power view of the edge of an ulcer at day 12. There is marked histological similarity to a human peptic ulcer. In the ulcer area granulation tissue has replaced the muscle coats and submucosa. The muscularis mucosae shows no evidence of regeneration. The liver is adherent to the serosa (H & E x 25).

mucosae or of a basement membrane.

The sequence of epithelial healing in these ulcers was in general agreement with that found by Williams[14] who studied superficial lesions in various laboratory animals, and by Myhre[10] who studied lesions in the rat. The epithelial coverage occurred at a rate of approximately 2 mm. a week which is in agreement with Ferguson's finding of 2 mm. per week mucosal regeneration in the dog.[4] The early contracture of the defects due to muscle contraction which Ferguson observed following surgical removal of the mucosa, was not found in this study. The reason for this was probably that the defect created in this study remained at first filled with tissue, albeit dead tissue. We did however find histological changes suggesting that early in the healing phase contraction of the muscularis mucosae and muscular coats tended to decrease the size of the mucosal and serosal portions of the defect. The main reduction in the size of the defect resulted from later contraction of the newly produced collagen.

It is of interest that after 60 days, by which time mucosal regeneration was complete and the granulation tissue in the gastric defect had been converted into dense scar tissue, microscopic mucosal erosions were found in a number of the healed

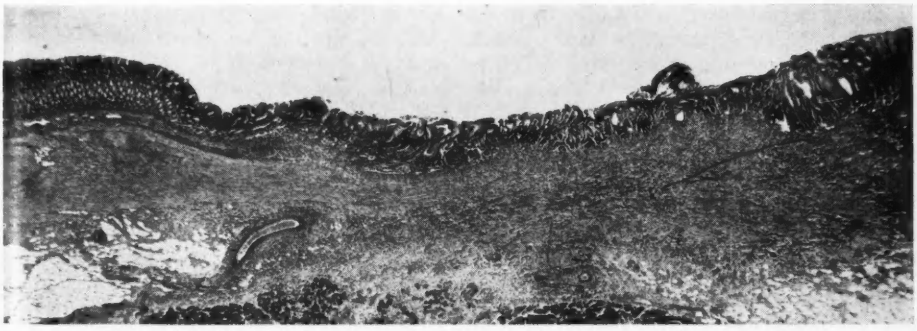


Fig. 7.—Day 21. The ulcer is completely covered by a regenerated mucosa. The muscularis mucosae is absent so that the reg nerated mucosa rests directly on the connective tissue base (H & E x 20).

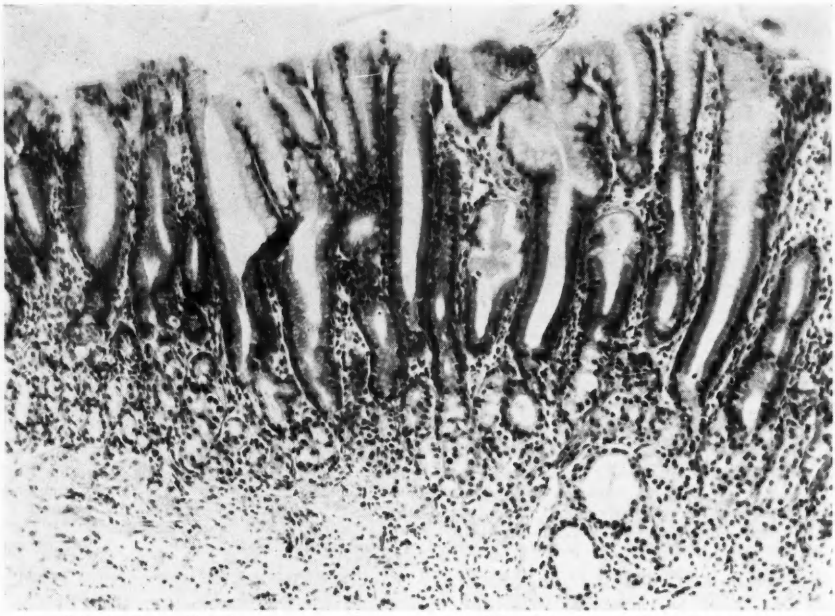


Fig. 8.—Day 28. The regenerated mucosa resembles that seen in the pyloric part of the stomach. The superficial epithelium and that of the foveolae are mucus secreting and are similar to the surface epithelium of the undamaged mucosa (H & E x 125).

ulcers. Spontaneous erosions and ulcerations in the rat's gastric mucosa are extremely rare.[14] This rarity, plus the fact that the erosions were invariably located in the middle of a healed ulcer area, suggests a definite relationship to the healed ulcer. It is our feeling that these mucosal erosions are related to the fact that the regenerated gastric mucosa lies directly on a scar tissue base without the normal interposition of a muscularis mucosae. In the early stages of healing, the tissue supporting the regenerated mucosa is a young pliable type of connective tissue which, with aging, becomes dense, rigid, collagenous fibrous tissue. By 60 days, the mucosa is supported by a dense scar tissue wall which lacks the usual protective mobility provided by the muscularis mucosae and so it is probably more liable to mechanical injury than is the normal mucosa.

Microscopic mucosal erosions were found starting from day 60 up until the termination of the experiment at day 100. We are unable to comment on the rate of healing of these on the basis of the material available for study. There would be reason to expect the healing process to be slower because of the underlying poorly vascularized, dense scar tissue from which the healing process has to be initiated. It has been suggested[2, 7] that the low vascularity and resulting ischæmic state of the fibrosed ulcer floor in human chronic peptic ulcers, is a factor contributing to the delay in healing of these ulcers. In the rat, although a healed penetrating ulcer of the fundus appears prone to spontaneous superficial mucosal re-ulceration, these were never found to penetrate to any degree, and did not result in anything resembling a chronic peptic ulcer. Thus it appears that factors other than mere mucosal ulceration of a healed penetrating ulcer are necessary for the production of a chronic peptic ulcer.

Summary and Conclusions

The healing of a standardized penetrating gastric ulcer was studied in a series of 300 rats. The healing process was found to be remarkably constant so that it was possible to predict the age of the ulcer on

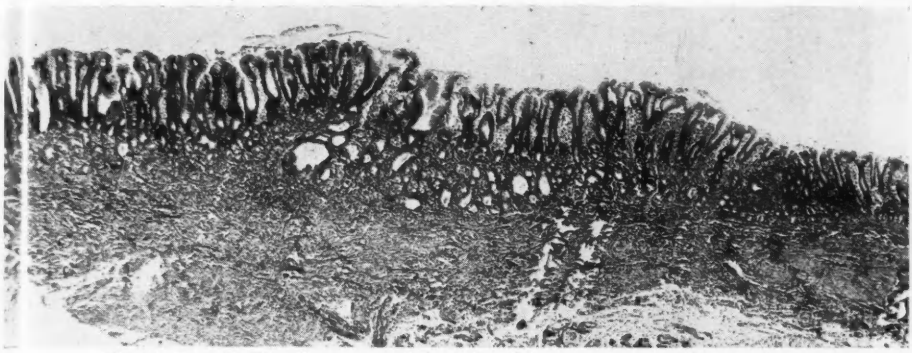


Fig. 9.—Day 40. The ulcers are well healed and less conspicuous. The granulation tissue of the wall has been converted to fibrous connective tissue. The ulcer site has contracted to a considerable degree (H & E x 25).

histological grounds, with great accuracy. This constancy allows for an accurate evaluation of the effects of physiological and pharmacological factors on the healing of this type of ulcer in the rat.

The base of the ulcer was formed by granulation tissue which, with aging, was converted into dense scar tissue. Concomitantly, the surface became covered by regenerated epithelium which with progressive differentiation showed a striking resemblance to normal gastric mucosa. The muscularis mucosae and muscle layers failed to regenerate.

Most of the ulcers were healed by three weeks and all were healed at four weeks. The ulcers remained healed until 60 days when centrally located areas of mucosal erosion occurred. These never penetrated to any depth. It is suggested that the healed ulcer site suffers mechanical injury more readily than the normal mucosa since it lacks a muscularis mucosae and is supported by rigid scar tissue.

Although healed penetrating ulcers in the fundus of the rat's stomach are prone to spontaneous mucosal erosion, this does not result in penetrating ulceration. In the rat, it appears that factors additional to mucosal erosion of a healed ulcer are necessary for the production of a chronic ulcer similar to a human peptic ulcer.

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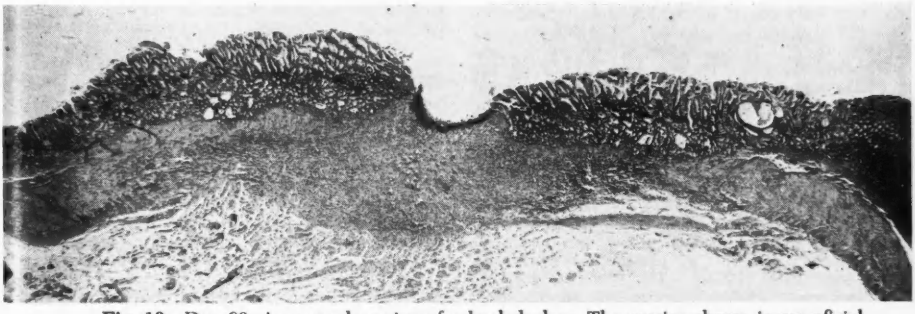


Fig. 10.—Day 60. A mucosal erosion of a healed ulcer. The erosion shown is superficial, shallow, and located in the central part of the healed ulcer. The healed area of the original ulcer consists of dense connective tissue (H & E x 20).

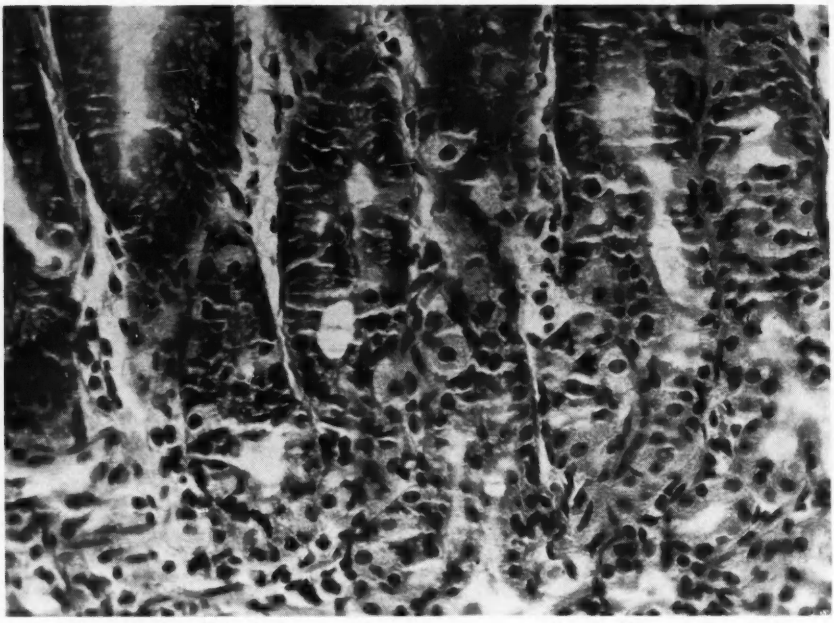


Fig. 11.—Day 100. High power view showing parietal cell differentiation in the regenerated mucosa (H & E x 400).

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Résumé

La pathogénèse de l'ulcère gastrique chez l'homme est basée sur trois facteurs: 1. ceux créant l'ulcération, 2. ceux localisant cette ulcération et 3. ceux qui entrent en jeu dans la guérison éventuelle de l'ulcération. Les auteurs étudient ce problème de guérison des ulcères dans une expérience comprenant 300 rats. Les détails expérimentaux et histologiques sont donnés.

Le processus de guérison des ulcères gastriques pénétrants semble être remarquablement constant, à tel point qu'on peut prédire l'âge de l'ulcus par son image histologique. Du moins chez le rat. Dans le processus de guérison, la base de l'ulcère est formée d'un maille de tissu de granulation qui plus tard se transforme en tissu fibreux dense. Entre temps, la surface de l'ulcère se couvre d'un epithélium régénératif qui, petit à petit, prend l'image de la muqueuse normale. A noter que la muscularis mucosae ainsi que la musculeuse font défaut dans cette guérison. Au bout de quatre semaines tous les ulcères expérimentaux se guérissent.

Toutefois, vers le 60ème jour, quelques-uns de ces ulcères montrent une récidive sous forme d'une érosion centrale de la muqueuse régénérée. Les auteurs ont constaté que les ulcères guéris sont plus susceptibles de lésion que la muqueuse normale, car il manque de la muscularis mucosae. Mais cette récidive chez le rat ne semble pas aboutir à l'ulcère chronique, ce qui est souvent le cas chez l'homme. Il apparait donc qu'il faut des facteurs autres qu'une ré-ulcération d'un ulcère guéri pour qu'on ait un ulcère chronique tel qu'on le trouve chez l'homme.

CANADIAN JOURNAL OF SURGERY

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Manuscripts

Manuscripts of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text and should be set out in accordance with the *Cumulative Index Medicus* abbreviation of journal name and general style. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. References to books should include in order: author's name; title of book; title of publishing house; city of publication; number of edition (e.g., 2nd ed.); year of publication.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10" x 8". Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

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It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

The 30th Annual Meeting of the College will be held at the Chateau Laurier Hotel in Ottawa from January 19 to 21, 1961. An excellent program is promised with four sessions of scientific papers to be presented in the Division of Surgery and three in the Section of Obstetrics and Gynæcology. In addition there will be a combined session of the Divisions of Medicine and Surgery dealing with the medical and surgical aspects of cardiovascular disease.

The Annual Lecture in Surgery will be delivered by Dr. Walter C. MacKenzie, Professor of Surgery and Dean of the Faculty of Medicine at the University of Alberta. Dr. MacKenzie's topic will be "Surgery of the pancreas".

The 1961 Medallist of the Royal College of Surgeons of Canada is Dr. John S. Speakman of Toronto. Dr. Speakman's winning essay entitled "The structure of the trabecular meshwork and corneal endothelium in relation to the problem of resistance to outflow in open angle glaucoma" will be presented at the meeting.

The Gordon Richards Memorial Lecture, offered annually by the Ontario Cancer Treatment and Research Foundation to a major scientific or medical meeting in Canada, will be given at the 1961 Annual Meeting of the College. This is the first occasion on which this lecture has been given at a Royal College meeting. The lecturer will be Dr. George E. Moore, Director of the Roswell Park Memorial Institute, Buffalo, N.Y., who will speak on the topic "Tumour cells and their spread".

On Friday and Saturday morning, January 20 and 21 respectively, two symposia of wide general interest to all Fellows of the College will be presented. One of these is entitled "Some interesting aspects of environmental medicine" and will deal with the medical problems of human adaptation to life or survival in environments of arctic cold, the underwater depths and outer space. Topic of the other symposium is "The irradiation hazard in Canada".

A special highlight of the meeting in Ottawa will be the official opening of the new College headquarters building by The Right Honourable John G. Diefenbaker, Prime Minister of Canada. This event will take place on Thursday evening, January 19.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary
December 2, 1960.

FORTHCOMING MEETINGS

UNIVERSITY OF TORONTO POSTGRADUATE COURSE IN OTO-LARYNGOLOGY

A graduate course in oto-laryngology will be presented by the Staff of the Department of Oto-laryngology on May 11, 12 and 13, 1961. They will be assisted by two distinguished guests, Dr. Philip E. Meltzer, Professor of Oto-laryngology, Harvard Medical School, and Chief of Oto-laryngology, Massachusetts Eye and Ear Infirmary, and Dr. W. G. Hemenway, Department of Oto-laryngology, University of Chicago.

The first session will begin in the afternoon of May 11, in the Royal York Hotel, Toronto, in association with the Section of Oto-laryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss, and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of responsibilities of this specialty in their management.

The fee for the course will be $40, and will include a complimentary dinner.

All enquiries should be addressed to the Director, Division of Postgraduate Medical Education, University of Toronto.

REFRESHER COURSE IN EYE SURGERY —UNIVERSITY OF TORONTO

The Faculty of Medicine, University of Toronto, will hold a Refresher Course in Eye Surgery from March 20 to 22, 1961. The instruction will consist of lectures, operative

sessions and a special symposium on cataract surgery.

Dr. Robert N. Shaffer, University of California, San Francisco, and Mr. B. W. Rycroft, F.R.C.S., London, England, will be guest surgeons. The staff of the Department of Ophthalmology will contribute extensively.

The course will be limited to 50 members and is open to eye, ear, nose and throat specialists. Application should be made to the Director, Division of Postgraduate Medical Education, Faculty of Medicine, University of Toronto, Toronto 5, Ontario, Canada, before January 31, 1961.

On March 18 there will be a Departmental Research meeting and Dr. H. M. Burian, University of Iowa, will be guest speaker. Members of the Eye Surgery Course are invited to attend.

NEWS FROM THE AMERICAN COLLEGE OF SURGEONS

At the 46th Annual Clinical Congress of the American College of Surgeons, held in San Francisco from October 10-14, 1960, approximately 1175 surgeons were inducted as new Fellows of the American College of Surgeons in cap-and-gown ceremonies closing the annual five-day Clinical Congress of the world's largest organization of surgeons. The American College of Surgeons, founded in 1913 to establish standards of competency and character for specialists in surgery, has grown in 47 years from a founding group of 450 to a total membership of approximately 24,000.

Fellowship, entitling the recipient to the designation "F.A.C.S.," following his name, is awarded to doctors who fulfil comprehensive requirements for acceptable medical education and advanced training as specialists in one or another of the branches of surgery, and who give evidence of good moral character and ethical practice.

Those receiving this distinction from Canada at the 1960 Convocation are listed below. Biographical material may be obtained from the physician's local medical society, his hospital or his office.

ALBERTA: Charles F. McCulloch and Wallace E. Mydland, Calgary, Morris Besney and Walter A. Strutz, Edmonton.

BRITISH COLUMBIA: Darrell F. Osborne, Kamloops. C. Richard Neve, Kimberley. Kenneth S. Weaver, Port Alberni. John A. Elliot, Duncan E. Govan and Gerald E. Howe, Vancouver.

MANITOBA: Charles M. Burns, Rankin K. Hay and John T. Macdougall, Winnipeg.

NEW BRUNSWICK: Claude Gaudreau, Edmunston.

NEWFOUNDLAND: Donald Cant, Corner Brook.

NOVA SCOTIA: George McK. Saunders, Amherst. Ian MacKenzie and H. Harold Tucker, Halifax.

ONTARIO: William S. M. Monk, Bracebridge. John B. Spence, Fort William. Walter A. Scott, Guelph. Charles C. Hopmans, Stephen E. O'Brien, Gerald J. Quigley and Richard T. Weaver, Hamilton. William R. Ghent, Kingston. Howard S. Cameron, Donald A. MacKenzie, Robert M. McFarlane and Jack H. Walters, London. Carstairs C. Gardner, Oshawa. Wallace B. Shute, Ottawa. Leslie J. Calvert, Peterborough. Robert R. Mutrie, Port Arthur. Getchel D. Williams, Port Credit. Gilles Helie and Kenneth A. Ward, Sudbury. Alan A. Bassett, James A. McIntyre, Robert I. Mitchell and J. Edward Mullens, Toronto.

QUEBEC: Laurent C. Archambault, Léon Beique, Michel J. Berard, G. Roger Champoux, Jean-Paul Cholette, Anthony R. C. Dobell, J. Cartier Giroux, Roger La Pointe, Patrick Madore, J. Maurice Parent, Yves Prevost, Roland Simard, R. J. Viger, Alexander M. Wright and M. H. Vincent Young, Montreal. Robert Cote, Quebec City. Wu Lou, Ste-Foy. Jacques E. Phaneuf and Paul Phaneuf, St. Jean. Maurice J. F. Chretien, Shawinigan. Pierre Grondin and René Matteau, Trois Rivières. Walter C. Lloyd-Smith, Westmount.

SASKATCHEWAN: Harold W. Estrey, Saskatoon.

NEW YORK UNIVERSITY MEDICAL CENTER COURSE IN SURGERY OF THE HAND

A five-day full-time course on surgery of the hand will be offered by the New York University Medical Center from March 6-11, 1961, under the direction of William T. Medl, F.A.C.S., and Thomas D. Rees, F.A.C.S. The course is designed mainly

for general, orthopædic, plastic and industrial surgeons.

The sessions will consist of practical demonstrations, lectures and panel discussions by members of the New York University faculty and a visiting faculty of eminent specialists in surgery of the hand. All facets of modern hand surgery, including anatomy, physiology, diagnosis, surgical techniques and management of hand lesions will be covered.

The guest faculty will include: Robert A. Chase, Yale University School of Medicine; Martin A. Entin, McGill University Faculty of Medicine; Jerome Gelb, Kessler Institute; J. William Littler, Columbia University College of Physicians and Surgeons; Erle A. Peacock, Jr., University of North Carolina School of Medicine; Lee Ramsay Straub and T. Campbell Thompson, Cornell University Medical College; Cdr. William C. Trier, M.C., U.S.N., Philadelphia Naval Hospital, and William L. White, University of Pittsburgh School of Medicine.

The class is limited to 40 participants and the tuition is $125. Applicants may obtain further information from the office of the Associate Dean, New York University Post-Graduate Medical School, 550 First Avenue, New York 16, N.Y.

FILM CONTEST AMERICAN COLLEGE OF CHEST PHYSICIANS

The Committee on motion pictures of the American College of Chest Physicians is interested in learning about new films on diseases of the chest (heart and/or lungs) for possible presentation at the 27th Annual Meeting of the College in New York City, from June 22-26, 1961.

Films accepted for presentation in the annual motion picture program are eligible for the 1961 film contest. The committee is also interested in reviewing new motion pictures for inclusion in the Approved Film List of the American College of Chest Physicians.

Physicians are invited to forward all pertinent information concerning their films to Dr. Paul H. Holinger, Chairman, Committee on Motion Pictures, American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

Winners of the 1960 College film contest are:

First Prize. Michael E. DeBakey and George C. Morris, Jr., Department of Surgery, Baylor University College of Medicine, Houston, Texas.
"Fusiform aneurysm of aortic arch, resection and replacement with Dacron graft"

Special Award. United States Air Force, Robert B. Stonehill, Lt. Col., M.C., U.S.A.F. Technical Adviser, Lackland Air Force Base, Texas.
"Air travel and the cardiopulmonary patient."

Honorable Mention. Chuzo Nagaishi, Tuberculosis Research Institute, Kyoto University, Kyoto, Japan.
"Direct intracavitary insufflation with chemotherapeutic agent using Metra's catheter via tracheo-bronchial route."

Honorable Mention. Theodore H. Noehren, University of Buffalo School of Medicine and Buffalo General Hospital, Buffalo, New York.
"Intermittent positive pressure breathing (IPPB/I)—its clinical application and administration."

AMERICAN COLLEGE OF SURGEONS 1961 Meetings

A sectional meeting will be held at the Hotel Dinkler-Tutwiler, Birmingham, Alabama, on January 16, 17 and 18, 1961. Dr. Arthur I. Chenowith of Birmingham is local Chairman.

A sectional meeting will be held at Hotels Del Prado, Reforma, Vista Hermosa, El Presidente, Alffer, Continental Hilton, Mexico City, Mexico, from January 23-26, inclusive. Dr. Gustavo Baz Prada is Honorary Chairman. Descriptive literature about this meeting, hotel and transportation facilities, pre- and post-meeting tours and advance registration forms may be obtained from the College headquarters.

From April 6-8, inclusive, a sectional meeting will be held at the Fort Garry Hotel, Winnipeg, Manitoba. Dr. Kenneth R. Trueman, Winnipeg, is local Chairman.

All Sectional Meetings are under the General Supervision of Dr. H. Prather

Saunders, Associate Director, American College of Surgeons. Information may be had on application to Dr. William E. Adams, Secretary, American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.

The 47th Annual Clinical Congress of the AMA will be held in Chicago, Illinois, from October 2-6, 1961.

AMERICAN COLLEGE OF SURGEONS

A four-day meeting for surgeons and nurses will be held in Philadelphia from March 6-9, 1961.

Surgeons and graduate nurses, and related medical personnel from all parts of the country are invited to attend this annual Sectional Meeting of the American College of Surgeons. Headquarters will be the Bellevue Stratford, Ben Franklin and Sylvania Hotels, with some sessions scheduled at leading hospitals in the city.

In length and scope this scientific meeting approaches that of the annual Clinical Congress of the College. The program will include hospital clinics, panel discussions, symposia, scientific papers, industrial exhibits, and medical motion pictures in general surgery sessions and in the specialties of obstetrics and gynæcology, ophthalmology, otolaryngology, urology, orthopædic surgery, plastic surgery, pædiatric surgery and thoracic surgery.

Nurses from Canada and all of the United States are invited to attend the meeting, the only joint nurse-doctor program scheduled by the College during 1961. Nurses and doctors will share the platform in team discussions of current problems.

Further information may be obtained from Robert S. Myers, M.D., F.A.C.S., Public Relations Department, American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Prophylaxie et Traitement des Escarres de Décubitus, par R. Vilain; préface du Dr. R. Guellette. 119 pp. Illust. Expansion Scientifique Française, Paris. $9.75.

The Case Reports and Autopsy Records of Ambroise Pare. Translated from J. P. Malgaigne's "Œuvres Complètes d'Ambroise Paré", Paris 1840. Compiled and edited by Wallace B. Hamby, M.D., F.A.C.S., Department of Neurological Surgery, Cleveland Clinic, Cleveland, Ohio. 214 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.25.

Chemical Osteosynthesis in Orthopædic Surgery. Michael P. Mandarino, M.D., Associate, Department of Orthopædic Surgery, Hahnemann Medical College and Hospital. 72 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

Demonstrations of Physical Signs in Clinical Surgery. Hamilton Bailey, F.R.C.S., F.A.C.S., F.R.S.E. Emeritus Surgeon, Royal Northern Hospital, London., Consulting Surgeon, Italian Hospital, General Surgeon, Metropolitan Ear, Nose and Throat Hospital, Vice-President, International College of Surgeons. 13th edition. 928 pp. Illust. John Wright & Sons, Ltd., Bristol, England; The Macmillan Company of Canada Limited, 1960. $12.75.

Electroencephalography in Anesthesiology. Albert Faulconer, Jr., M.D., M.S., Head, Section of Anesthesiology, Mayo Clinic, and Reginald G. Bickford, M.B., Ch.B., M.R.C.P., Consultant, Section of Physiology, Mayo Clinic. 90 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.25.

Les Entretiens de Bichat 1960. Chirurgie-spécialités. (Colloquium of Bichat, Surgery and Specialties, 1960). Edited by R. Gueulette, J. Hepp, Y.-J. Longuet and M. Roux, chirurgiens de l'Hôpital Bichat. 557 pp. Expansion Scientifique Française, 1960.

Gynecological Urology. Compiled and edited by Abdel Fattah Youssef, M.B., D.S., D.G.O., M.Ch., F.I.C.S. Lecturer in Obstetrics, Cairo University. 893 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $24.25.

Hypothermia for the Neurosurgical Patient. Antonio Boba, M.D., Associate Professor of Anesthesiology, the Albany Medical College of Union University, Albany, N.Y. 124 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.50.

Klinische Chirurgie fur die Praxis. In vier Banden. Band III. Lieferung 2. (Clinical Practice of Surgery. In four volumes. Vol. III, Part 2). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 360 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $9.50.

Memory, Learning and Language. The Physical Basis of Mind. Edited by William Feindel. University of Saskatchewan Jubilee Symposium, 1959. 69 pp. Illust. University of Toronto Press, 1960. $2.00.

Modern Trends in Cardiac Surgery. H. R. S. Harley, M.S., F.R.C.S., Consultant thoracic surgeon, United Cardiff Hospitals and Welsh Regional Hospital Board. 292 pp. Illust. Butterworth & Co. Ltd., London and Toronto, 1960. $14.00.

Neurology Simplified. David Joseph LaFia, M.D., Assistant in Neurosurgery, Jefferson Medical College and Hospital, Philadelphia, Pennsylvania. Foreword by A. Earl Walker, M.D., The Johns Hopkins University School of Medicine. 174 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.50.

Non-penetrating Injuries of the Abdomen. Robert H. Kennedy, Consulting Surgeon, Beekman-Downtown, Bellevue and University Hospitals, New York City. Edited by Lester R. Dragstedt, Research Professor of Surgery, the University of Florida, Gainesville, Florida. 121 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.25.

Procedures in Vascular Surgery. Richard Warren, M.D., Clinical Professor of Surgery, Harvard Medical School. 211 pp. Illust. Little, Brown and Company, Boston and Toronto, 1960. $12.00.

Radiology as a Diagnostic Aid in Clinical Surgery. Howard Middlemiss, M.D., F.F.R., D.M.R.D., Director of Radiology, United Bristol Hospitals, Lecturer-in-Charge, Department of Radiodiagnosis, University of Bristol, England. 150 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $9.00.

Selected Papers. Sir Geoffrey Jefferson. 563 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $26.50.

A Short History of Obstetrics and Gynecology. Theodore Cianfrani, Associate Professor of Obstetrics and Gynecology at the Graduate School of Medicine, University of Pennsylvania, Philadelphia. 449 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $13.75.

Stress and Psychiatric Disorder. Edited by J. M. Tanner, M.D., D.Sc., Secretary of Research Committee, Mental Health Research Fund. 151 pp. Illust. Blackwell Scientific Publications, Oxford; The Ryerson Press, Toronto, 1960. $7.55.

The Surgeon's Glove. Justine Randers-Pehrson, M.A. 95 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

Surgical Diseases of the Pancreas. John M. Howard, M.D., F.A.C.S., Department of Surgery, Hahnemann Medical College, and Surgeon-in-Chief, Hahnemann Hospital, Consultant in Surgery, Walter Reed Army Medical Center; and George L. Jordan, Jr., M.D., M.S., F.A.C.S., Associate Professor of Surgery, Baylor University College of Medicine. 607 pp. Illust. J. P. Lippincott Company, Philadelphia and Montreal, 1960. $20.00.

Surgical Treatment of Unequal Extremities. Charles Weer Goff, M.D., Associate Clinical Professor of Orthopædic Surgery and Lecturer in Anatomy, Yale University School of Medicine. Member, La Société Internationale de Chirurgie Orthopédique et de Traumatologie. 184 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $12.00.

Thymectomy for Myasthenia Gravis. A record of experiences at the Massachusetts General Hospital. Henry R. Viets, M.D., Lecturer on Neurology, Emeritus, Harvard Medical School, and Robert S. Schwab, M.D., Assistant Clinical Professor of Neurology, Harvard Medical School. 143 pp. Illust., Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.75.

The Tonsils and Adenoids in Childhood. Donald F. Proctor, M.D., Associate Professor of Laryngology and Otology, The Johns Hopkins University School of Medicine. 70 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $8.25.

BOOK REVIEWS

MEDICAL, SURGICAL AND GYNECOLOGICAL COMPLICATIONS OF PREGNANCY. The Staff of the Mount Sinai Hospital, New York. Edited by Alan F. Guttmacher, M.D., Obstetrician and Gynecologist-in-Chief, The Mount Sinai Hospital, New York, and Joseph J. Rovinsky, M.D., Assistant Attending Obstetrician and Gynecologist, The Mount Sinai Hospital, New York. 619 pp. Illust. The Williams & Wilkins Company, Baltimore, Maryland, 1960. $16.50.

The attending staff of Mount Sinai Hospital under the editorship of Alan F. Guttmacher and Joseph J. Rovinsky have presented an interesting and informative volume on the medical, surgical and gynæcological complications of pregnancy. Each chapter is concise, well written, and covers the literature up to date. The experiences of each individual author are included with special reference to the cases that have been seen at the Mount Sinai Hospital. Physiology, pathogenesis and treatment of each complication are noted.

The long list of references at the end of each chapter is particularly valuable, and this book will be of great value to both the specialist and general practitioner.

CLINICAL ORTHOPÆDICS. Editor-in-chief Anthony DePalma, with the assistance of the Associate Editors, the Board of Advisory Editors, and the Board of Corresponding Editors. Number 16. 315 pp. Illust. J. P. Lippincott Company, Philadelphia and Montreal, 1960. $7.50.

The Sixteenth Volume in the series of Clinical Orthopædics supplies stimulating, theoretical and practical information on the treatment of

(Continued on page 257)

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(Continued from page 254)

many of the commoner lower limb and foot problems. Much valuable information is presented in a clear and concise manner. The standard procedures are well illustrated and the references are adequate. Some controversial procedures are described which are worthy of further study and evaluation.

The young orthopædic surgeon will find this volume, like the preceding ones, an invaluable aid in the assessment and treatment of many common orthopædic problems. Many of the articles will appeal not only to the orthopædic surgeon, but to all those interested in the postural and static deformities of the limbs.

CLINICAL TROPICAL DISEASES. A. R. D. Adams and B. G. Maegraith, Tropical School of Medicine, Liverpool, England. 2nd edition. 340 pp. Illust. Blackwell Scientific Publications, Oxford; The Ryerson Press, Toronto, 1960. $12.50.

When this small textbook was first published seven years ago, it was welcomed both for its simplicity of approach and the value of the material which had been selected. The same format has been followed in this second edition, with inevitably some enlargement, due to the gains of knowledge over the past few years, of this ever widening subject. This is particularly evident in the emphasis that has been given to the nutritional diseases and those due to physical effects.

The arrangement of diseases in alphabetical order facilitates easy reference, a matter of prime importance in a book of this type, which is obviously a handbook in the widest sense. There are perhaps one or two obvious omissions, such as the lack of description of the methods of control of disease, but as the authors pointed out in their first edition, this was not the essential purpose of the book, neither was it to provide more than the briefest information on both organisms and vectors. They have adhered to this policy in the present edition, and probably rightly so, because such information is available elsewhere and is not within their terms of reference.

There are a few masterly presentations, such as the description of blackwater fever, their emphasis of established treatment of amœbiasis and the succinct description of clinical effects of heat and light.

Criticisms are of a more personal kind in that the binding of the book is inferior and probably will not stand up to a tropical climate for very long.

There is no doubt that this book will be found as useful as its first edition and could well find a place on many hospital library shelves in this country, because so-called tropical diseases are becoming increasingly prevalent in temperate climates, owing to the rapidity of travel both of vacationers and immigrants.

EXPOSES D'ANESTHESIOLOGIE. A l'usage des practiciens et des étudiants. P. Huguenard et P. Jaquenoud. Première série. 218 pp. Illust. Mason & Cie, Paris. 23NF.

EXPOSES D'ANESTHESIOLOGIE. A l'usage des practiciens et des étudiants, P. Huguenard et P. Jaquenoud. Deuxième série. 274 pp. Illust. Masson & Cie, Paris. 23NF.

These publications provide information of value for the medical practitioner, the medical student and the anæsthetist. They provide physiological and pharmacological aspects of anæsthesia and problems related to this field. Helpful hints on the technique of answering examination questions are provided for the candidate preparing for specialist qualification. Many of the aspects of anæsthesia not to be found in other books on this subject are discussed in detail in these publications.

INTRASPINAL TUMORS OF CHILDHOOD. Robert W. Rand, Assistant Professor of Neurological Surgery, University of California School of Medicine, Los Angeles, and Carl W. Rand, Emeritus Professor of Neurological Surgery, University of Southern California School of Medicine, Los Angeles. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $18.25.

The basis for this monograph is the authors' material of 72 cases, a not inconsiderable series when viewed against the 214 cases which, according to Hamby, was the total that had appeared in the literature up to 1942. The cases are arranged according to their pathological types and while each chapter is largely taken up with case reports (as is proper in a book of this nature) there is also full discussion of the problems involved, pathological, diagnostic and therapeutic. We are thankful that the authors have been so painstaking in recording their extensive experience in this branch of surgery. The illustrations and production are excellent, marred only by the inept advertising remarks of the publisher on the dust cover face.

One of the disadvantages of working in our nomadic society is that an adequate long term follow-up study is very difficult to achieve. Or the net is cast too late in one's career to keep all the cases captive. An admirable feature of this book is the thoroughness of the post-operative record. We are not often let down with an "untraced", or with the anticlimax of a follow-up too short to be of much significance.

Many of the cases in the glioma group received radiation but whether or not it was effective in retarding the progress of the disease is still unknown. This is one of the fields of therapy in which we are still wandering blindly; a well controlled study (if that is

possible) of brain and cord gliomas is badly needed.

EXPERIENCES WITH CONGENITAL BILIARY ATRESIA. Julian A. Sterling, M.D., ScD., F.A.C.S., Senior Attending Surgeon, Albert Einstein Medical Centre, Assistant Professor of Surgery, Graduate School of Medicine, University of Pennsylvania. Publication No. 392, American Lecture Series. A Monograph in American Lectures in Abdominal Viscera. Edited by Lester R. Dragstedt, M.D. 68 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.00.

This book is a rare little gem, reflecting the ingenious courage, perseverance and surgical sagacity of the author and the wisdom of the publisher, both previously renowned.

In the preamble is a succinct classification of the usually fatal congenital biliary agenesis with the observation (histologically) that canaliculi and tiny bile ducts are usually present and the late stages resemble nodular cirrhosis; obstructive jaundice in the first month of life is probably due to atresia of the bile ducts.

The author describes the management of 10 patients and concludes that for success: (1) The liver must produce bile. (2) After the bile is *internally* drained from a hepatic lobule, the lobule is normal. (3) The silver drainage tubes into the liver must be anchored; and speculates that (4) liver drainage may aid the "delayed formation" of intrahepatic ducts.

Almost insurmountable difficulties and deficiencies were encountered in these patients, but tangible gratifying progress was made. Interspersed through the monograph are pertinent tables, diagrams, radiographs and photomicrographs. An extensive reference list is provided.

Like a true jewel the book is as durable on the surface as genuine inside. It should be in most large hospital libraries and read by every abdominal and general surgeon faced with the problem.

GLOSSARY OF WORDS AND PHRASES IN RADIOLOGY AND NUCLEAR MEDICINE. Lewis E. Etter, B.S., M.D., F.A.C.R., Professor of Radiology and Chief, Radiological Service, Western Psychiatric Institute and Falk Clinic School of Medicine, University of Pittsburgh. With a section on Suggested Terminology for Roentgenological Reports, devised by Drs. Fisher, Bovard and Bacon for the Pennsylvania Radiological Society. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $9.25.

This is a book of words and phrases commonly used in the field of radiology. It has been compiled as an aid to medical secretaries, x-ray technicians, radiologists in training, and other workers and writers on radiological subjects.

The glossary is extensive, without obvious omissions. In addition, there is a brief discussion of "Radiological Symbols and Semantics" and a very good compendium of suggested terminology for roentgenological reports.

This book, admirably conceived, falls somewhat short of its objectives owing to editorial and typographical errors. Unfortunately, the author did not follow the usual practice in dictionaries of listing the words in small letters, using capitals only for proper names. Since everything is capitalized in this book, the relatively uninitiated person to whom it is addressed is bound to find some confusion. (How could one tell whether March Fracture and Block Vertebra represented eponyms or not?) There are many misspellings of both simple words and proper names, and errors of fact occur in some definitions. Some adverbs are listed as adjectives. The sample roentgenological reports are generally good, but contain a minimum of such undesirable jargon as: "The gallbladder fails to visualize", and such and such "is believed to represent . . ."

This edition, although undoubtedly useful, can be recommended only with considerable restraint, as there are far too many minor errors for a first class work of reference. It is to be hoped that a new edition will soon be forthcoming, since careful editing and proof reading would make this an extremely worthwhile volume.

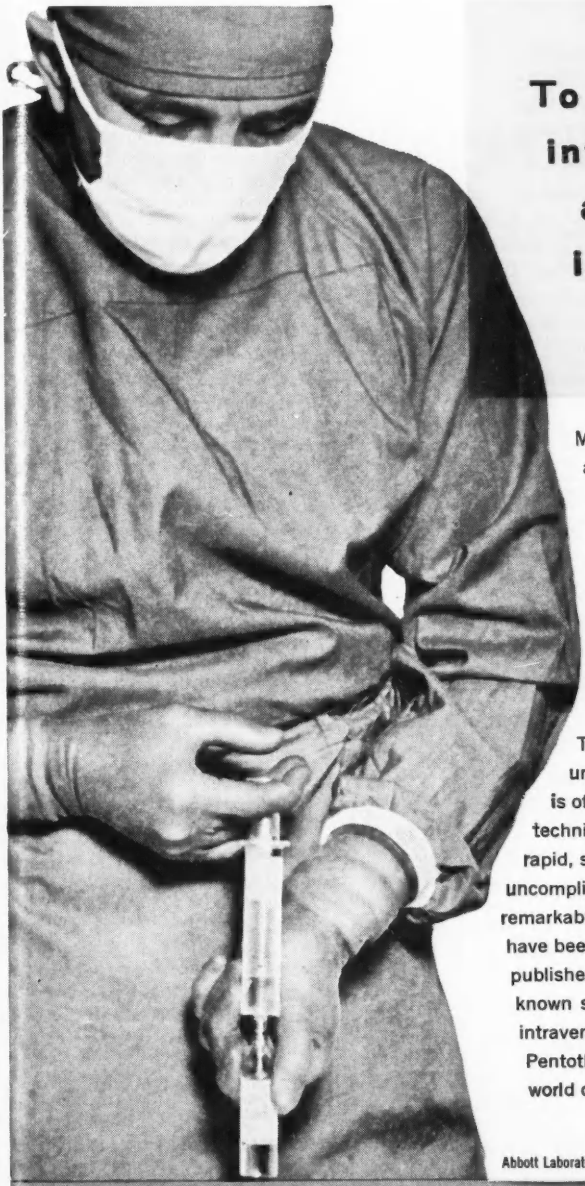
TRANSACTIONS AMERICAN SOCIETY FOR ARTIFICIAL INTERNAL ORGANS. Vol. VI. Edited by George E. Schreiner, M.D., and Associate Editors John F. Maher, M.D., Modestino Criscitiello, M.D. and Joan P. Ryan, Georgetown University Press for A.S.A.I.O. Copies may be ordered from Dr. George E. Schreiner, Department of Medicine, Georgetown University Hospital, Washington 7, D.C. $8.00.

This is a collection of the papers presented at the Sixth Annual Meeting of the Society (once referred to jocularly by one of its members as the society for open hearts and shutdown kidneys) held in April 1960, and of the sometimes too literally transcribed discussions which ensued. It has the merit of presenting the visual material utilized by the discussants, and the 10 minute time limit imposed on presentations makes for adequate discussions as well as for easy reading.

The papers presented logically divide themselves into three categories: (1) Hæmodialysis —the artificial kidney. (2) Pumps and Oxygenators. (3) Heart and heart valve replacements.

The first two of these are by now clinically *fait accompli*. The day of satisfactory prosthetic valves, particularly the aortic, may now be close at hand. Although some exciting work is now in progress on implantable artificial

(Continued on page 260)



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ANNOUNCEMENT

THE ALEXANDER GIBSON MEMORIAL LECTURESHIP—UNIVERSITY OF MANITOBA

The inaugural Alexander Gibson Memorial Lecture will be delivered by Sir Walter Mercer, Emeritus Professor of Orthopædic Surgery, University of Edinburgh, in the auditorium of the Medical College, Winnipeg, at 8.30 p.m. on January 24, 1961. The topic of the address will be "Edinburgh's Contribution to Medicine in America".

The late Dr. Alexander Gibson was formerly Professor of Anatomy, and later, Professor of Orthopædic Surgery at the University of Manitoba. Dr. Gibson's contributions to medicine in the fields of anatomy and orthopædics are widely recognized, and the University of Manitoba is proud to inaugurate this lecture series to commemorate his many outstanding services to the profession.

(Continued from page 258)

hearts, the time when a surgeon can replace a sick or defective heart with a synthetic substitute, would seem to be fairly remote.

Only a minority of the papers dealt with engineering problems and instrumental design and the majority were concerned with associated physiological and metabolic problems. This volume will provide interesting reading for those already engaged in investigation in these various fields but would not be recommended as a primer for those with only casual familiarity in these areas.

THE TRANSPARENCY OF THE CORNEA. A Symposium organized by The Council for International Organizations of Medical Sciences. Established under the joint auspices of UNESCO and WHO. Edited by Sir Stewart Duke-Elder and E. S. Perkins, Institute of Ophthalmology, London. 268 pp. Illust. Charles C. Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $12.00.

This book is the result of the Symposium on Transparency of the Cornea which was held in relationship to the 18th International Congress of Ophthalmology in Belgium in 1958. Twenty-two experts on the cornea, both clinicians and researchers, contributed. Sir Stewart Duke-Elder states that the purpose of the

MODERN TRENDS IN CARDIAC SURGERY

Edited by H. R. S. HARLEY, M.S., F.R.C.S., Consultant Thoracic Surgeon, United Cardiff Hospitals and Welsh Regional Hospital Board.

307 pages. Illustrated. $14.00 delivered.

This volume was produced because it was felt that the time was propitious for an expression of British thought and practice in cardiac surgery. The 22 contributors representative of surgical and medical thought in all parts of the country have combined to cover a wide variety of topics. After a historical introduction recording the rapid and dramatic advances made in the surgery of the heart and great vessels during the last twenty years, the subjects dealt with cover aspects of current interest, giving full attention to new techniques such as hypothermia, deep hypothermia and extra corporeal circulation.

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Symposium is to discuss the following problems: (1) Why is the normal cornea almost transparent? What is the physical basis of this property unique among adult mesodermal tissues? (2) How is the transparency maintained throughout life? (3) What do we know of the pathological factors that cause a loss of transparency? How can it be treated? (4) The question of transparency in corneal grafts.

The book covers in orderly fashion all aspects dealing with corneal transparency, beginning with a microscopic study of the layers of the cornea, accompanied by excellent photographs. Norman Ashton outlines the effect of swelling on the staining reactions of the cornea, and later in the book discusses the various thoughts on corneal vascularization. Dohlman, Maumenee, Paufique, Leigh and Franceschetti go into the question of corneal grafts and all the factors affecting their transparency.

This book is of particular value to those carrying out research projects on the cornea, but most ophthalmologists will find the discussions of these brilliant men most stimulating.

TETANUS. PROPHYLAXE UND THERAPIE. (Prophylaxis and Therapy of Tetanus). L. Eckmann. 128 pp. Illust. Benno Schwabe & Co. Verlag, Basle and Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $4.00.

Professor Nissen in his introduction to this little volume draws our attention to the fact that tetanus, even in peacetime, is still a much underestimated infection with a mortality of approximately 40%. Besides the tetanus acquired by trauma—the majority of cases—one still sees cases after abortion, confinement, and operations in the newborn.

Prophylaxis is emphasized, and after presenting the methods of passive and active immunization the author concludes from numerous experimental, clinical and statistical observations that the beneficial effect of passive immunization, in the form of heterologous antitoxin-serum, is questionable, and indeed immunization may often be harmful. The simultaneous use of serum plus toxoid is, however, advocated.

The therapy of the already developed infection is discussed in a separate chapter. This could be medical, mainly symptomatic treatment, or surgical, in the form of excision of the damaged tissues and tracheotomy. For serum therapy the use of antitoxin is recommended. One intravenous injection of 10,000 to 20,000 international units of antitoxin with calcium and antihistamine may be given.

The extensive list of references makes this excellent monograph particularly useful.

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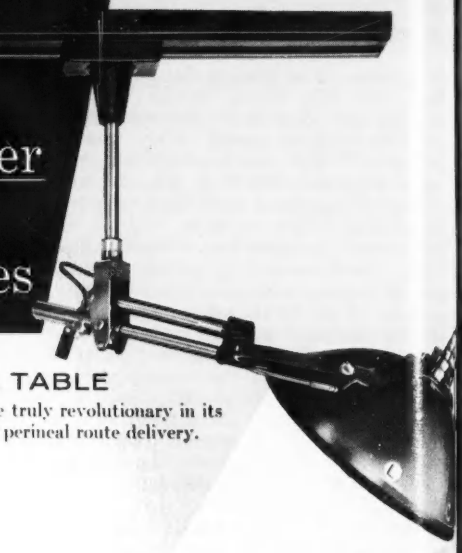
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HISTORY OF CANADIAN SURGERY

JOHN STEWART*

HAROLD L. SCAMMELL, M.D., *Halifax, N.S.*

It is now more than a quarter of a century since Dr. John Stewart died, yet his memory has remained as an inspiration to those who knew him, and his tradition has lived on in his native province and in the medical school to which he gave such exemplary service.

Dr Stewart was born on July 3, 1847, at St. George's Channel, Richmond County, Nova Scotia, where his father, the Reverend Murdoch Stewart, was the Presbyterian minister. He attended the Model School in Truro, Nova Scotia, and later the Provincial Normal College in the same town. After graduation he taught for one year in Sydney Academy, and then resolved to study medicine. He entered Dalhousie College for this purpose but because the future of medical education there was at that time uncertain, he transferred to Edinburgh. He graduated M.B., C.M., on October 6, 1877, at the age of 30.

Immediately before Stewart's graduation, Professor Joseph Lister had been invited to accept the Chair of Clinical Surgery at King's College Hospital, London. The story of events which preceded and followed this is part of well recorded medical history. Suffice it to say that Lister chose John Stewart as one of his house surgeons, familiar with his technique of antiseptic surgery, to go to London with him. In later years he used to recall vividly how he rode to Lister's first lecture in a cab, carrying with great care the culture tubes demonstrating the presence of living organisms. It was a day of days, and one on which the sun rose to give new light and hope to a suffering world.

If Stewart thought about his future at all at that time it would have seemed bright to any ambitious young man. True, Lister was not accepted in London with open arms or with immediate acknowledgment of the greatness of his discovery, but he had supreme faith in its value, a faith amply justified by its use. He was a son of a wealthy family; he could afford to wait a while. In the meantime, his house surgeons knew that they were standing with him on the brink of an epoch. All that was necessary was to work well and wait. Stewart, a man over six feet tall, handsome, dignified and of gentle bearing, could look forward to a career full of honours and achievement. Within a year, if such a dream existed, it vanished, at least for the moment. His father had died and family needs required his presence in Nova Scotia as head of the family. If he had regrets nobody knew of them. He returned to Canada and began to practise in Pictou where his brother was a lawyer.



Fig. 1.—Dr. John Stewart

Those were days of a great educational awakening in Pictou. The college, founded there in 1816 by Thomas McCulloch, D.D., had vanished and left in its place a struggling Academy to recall its former glory, which now seemed in revival. George Munro, a native of Pictou County had made a fortune as a publisher in New York, and, not forgetting his old school, he created a number of scholarships and bursaries available to its graduates, which enabled many to secure a

*C.B.E., M.B., C.M., (Edin.), F.R.C.S. (Edin.), LL.D (Edinburgh, Dalhousie and McGill).

university education. Students came to it from afar, the quality of its teaching became unsurpassed in the Maritime Provinces at that time, and its fame returned. Dr. John Stewart was deeply interested in the school and its students. He was single, he lived near the Academy and rejoiced in associating with the students and members of its faculty. He was a naturalist and his love of the beautiful countryside around Pictou made it easy for him to steal away from a busy practice for long walks with a friend. In later years he was regarded with some wonder as a botanist and ornithologist. Dr. Stewart's extensive and accurate knowledge in both of these sciences was gained almost unconsciously in the pleasant school of friendly and cultured associations.

Meanwhile his brother's family was growing and as he was unmarried, Dr. Stewart gave them the affection his own children would have won. He was an inspiring uncle. Football and lacrosse were popular games in Pictou in those days, and in these he excelled. The Pictou County Artillery Company was organized and he became one of its officers. As a younger man he had held a commission in the Third Regiment of Richmond County. As a surgeon he was gaining a wide reputation.

When Dr. Stewart began his practice there was but one general hospital in Nova Scotia, the Provincial and City Hospital in Halifax, which was over 100 miles from Pictou by road or rail, and much farther by water. As a result, and in keeping with good practice of that day, operations were done at home. If antiseptic surgery seems archaic in our day, it must be admitted that it lent itself admirably to the kitchen table era. Provided the surgeon had instruments and linen, the average household supplied all other needs but a few ounces of chloroform and a bottle of carbolic acid crystals. Stewart had taken a Lister spray from London and it is now at Dalhousie University. That took care of the operation field. Instruments were immersed in 1:20 carbolic acid. Hands were scrubbed and immersed from time to time in the same solution. Linen, sponges and sutures all went through the same process. Any inadequacies were taken care of by the acquired resistance of members of the household to their native bacteria. Chloroform, properly given, was a safe anesthetic. Dr. Stewart had been brought up in a school where speed and dexterity were highly valued. He made full use of these, but as he gained confidence in his developed methods he exhibited a wonderful degree of care and thoroughness. Fifty years after John Stewart began to practise in Pictou, I made the same attempt in the same town. There were men and women living to exhibit the honourable scars received at his hands and to attest to the greatness of his skill. At the same time there were physicians in practice who had worked with him during what we would now consider the pioneer days. One of these told me of a young man who had developed a tuberculous lesion of the hip joint with abscess formation. The narrator was asked by Dr. John Stewart to give the anesthetic. The room was prepared, the kitchen table draped and everything was in readiness. Before the anesthetic was begun, the surgeon gathered the family together in the kitchen and in a simple unaffected way told them what he was going to attempt and ended by asking God's help in the task before him. "Then", continued my informant, "we went into the improvised operating room. Dr. Stewart cut down on the head of the femur and curetted away the last bit of necrotic bone. He swabbed out the abscess track with a sponge which had been wrung out of carbolic acid solution; then he closed the incision without drainage, all of which took about two hours. The wound healed by first intention." I asked, "What became of the patient?" He replied, "You see that man plowing over on that hill; he is the man." Forty years had passed!

It was during the Pictou years that Stewart was an unwilling participan in an episode that caused him much discontent and uneasiness. In 1885, owing to a dispute over an appointment at the Provincial and City Hospital in Halifax, the entire visiting staff resigned. As it was the only teaching hospital then available, the Halifax Medical College decided to

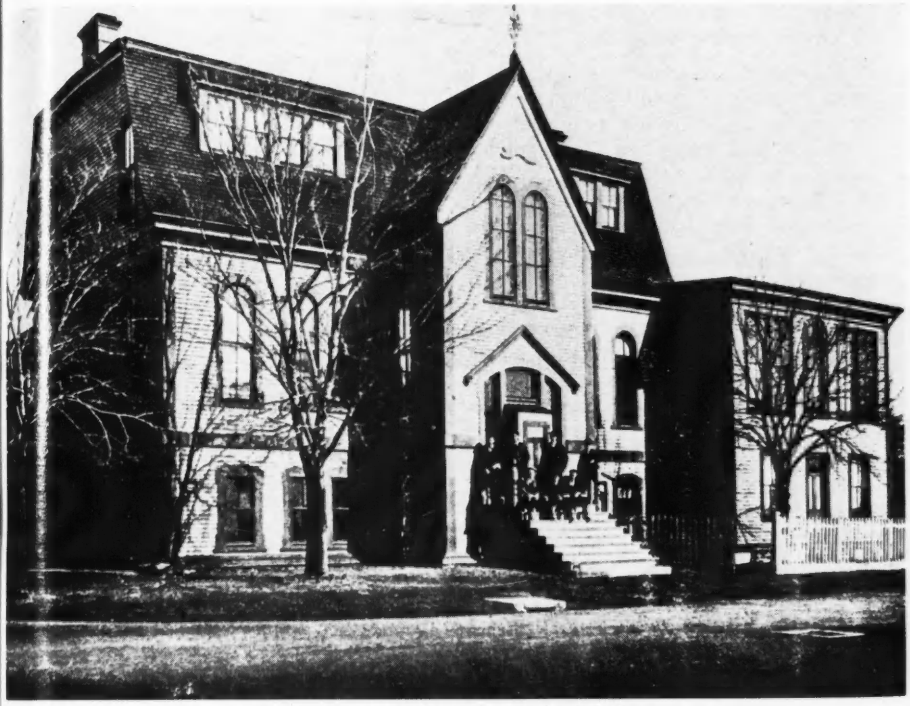


Fig. 2.—The original Halifax Medical College

suspend operations as well. The entire matter was fully investigated and administrative matters at the Hospital were the subject of severe public criticism. Since Dr. Stewart had a comprehensive knowledge of hospitals in both London and Edinburgh, as well as of some on the Continent, his opinion was sought and given. It was a time when feelings ran high, and no opinion was likely to meet with universal favour. In any event, his was a fair and honest appraisal of the situation as he saw it. Eventually a solution was reached which removed the hospital from joint ownership by the City of Halifax and the Province of Nova Scotia, and conferred its sole ownership upon the Province. The bed capacity was increased by 100, and the institution was renamed the Victoria General Hospital in 1887, Queen Victoria's jubilee year.

In the same year the Sisters of Charity, at the suggestion of Dr. Stewart, opened a small hospital on Barrington Street in Halifax which they named The Victoria Infirmary. It was the beginning of the present Halifax Infirmary.

By 1894, Dr. Stewart decided that there were no immediate prospects for the erection of a hospital in Pictou County, and that he could pursue a career of greater usefulness in the province's capital city. He was at once appointed to the Faculty of Medicine at Dalhousie University, but did not choose to become a member of the staff of the Victoria General Hospital. As long as he continued in active practice the greater part of his work was done at the Infirmary. By this time aseptic surgery was replacing antiseptic surgery. It was difficult for him to change from a method which he had seen at its birth and which he had used so successfully for many years of practice. There was also a feeling of transcendent devotion and loyalty to his old teacher Lord Lister, that made him loath to relinquish his methods. He changed slowly and reluctantly, and when called out of Halifax to operate, as occurred constantly, he

would revert to former techniques if the local circumstances permitted.

It is almost impossible today for a younger member of the medical profession to grasp the spirit which pervaded the practice of medicine and its evolving specialties in Nova Scotia from 1894 to 1914. Those who aspired to recognition in a special field had emerged, almost without exception, from a background of general practice. Sometimes the transfer to specialized practice was heralded by a period spent in one of the large centres in the United States, the British Isles or Germany; in other instances it would be founded upon local success and experience. In most instances the arrival of a new surgeon on the scene was not greeted with applause by his new associates. All were rugged individualists, accustomed to working alone. They were resourceful and aggressive. In Halifax, practice was highly competitive. The poor were treated, and well treated, free, and there were many poor. Consequently the paying patient was jealously watched. The "little books" on ethics were frequently consulted and unethical conduct was a common accusation levelled in official conclaves. Into this professional maelstrom came John Stewart. Where most others would have failed, he succeeded beyond the most sanguine hopes.

The reasons for this success were many. In the first place, his training and skill as a surgeon were first class. In the second place he had no desire to make money—a modest competence was all he asked. Finally, in every word and action, he was a man above the crowd. There was no malice in his being, and he was never known to speak unkindly of anyone. Though deeply religious, he did not force his opinions or beliefs on anybody but was a living example of the strength of his faith. Where his skill alone might have failed, his lack of self-seeking, and unassuming uprightness of character won. He remained stately and serene above his bickering associates who soon grew to trust and love him.

He was elected President of the Canadian Medical Association in 1905. In 1906 he was appointed to the Provincial M lical Board and later became its presidei . In 1913 he became Professor of Surge y at Dalhousie University.

Then, in 1914 came World War . In 1915 the Dalhousie University Num er 7 Stationary Hospital was organize for service overseas. In spite of his 67 ears, John Stewart was placed in comma l by unanimous consent. The unit re ched France early in 1916 and remained here until the end of the war. On July 3, 1917, the Hospital was at Arque, and on that day it was visited and inspected by King George V. His Majesty spent some hours there in conversation with the staff and Colonel Stewart, who afterwards said that it was only by a great effort of restraint that he refrained from telling the king that it was his seventieth birthday. In March 1918, he was transferred from his command to Canadian Army Medical Corps Headquarters in England as Consulting Surgeon.

On his return to Canada on January 6, 1919, Dr. Stewart was tendered a banquet of welcome by educational, business and professional men of Nova Scotia. On October 6, 1927, when he celebrated 50 years of practice he was again honoured at a banquet and presented with a purse of gold.



Fig. 3.—Lister spray which Dr. Stewart u d in his operations.

Following his return from overseas Dr. Stewart began to retire gracefully. In 1919 he was made Dean of the Faculty of Medicine at Dalhousie University, and the cares of an expanding school occupied much of his time. However, he was Consulting Surgeon at Camp Hill Hospital, and would from time to time see old friends in consultation, but his active days of practice were over.

Honours began to almost crowd upon him. He had retired from active service in the Army with the rank of Colonel and was made a Commander of the British Empire. In connection with the Lister Celebration in 1927, the Royal College of Surgeons of Edinburgh bestowed on him their Honorary Fellowship, which is the highest award the College is able to bestow and which is awarded very sparingly. He was also given the honorary degrees of Doctor of Law by both Dalhousie and McGill Universities. He was twice President of the Medical Society of Nova Scotia; once, as previously noted, of the Canadian Medical Association, and once of the Medical Council of Canada. He delivered the first Listerian Oration to the Canadian Medical Association in 1924, which revealed his unexcelled mastery of the English language.

In the spring of 1932 Dr. Stewart led his last medical class to graduation. A few days before, he had heard them subscribe to the Hippocratic Oath. It was an event to remember, the tall bearded figure unbent by his 85 years, standing before them. It must have recalled to his mind a similar scene in his own day. He told them in simple language of their obligations to themselves and to society. He ventured the hope that they were going out with some inspiration, to live full and useful lives. Then in tones which at times were almost a whisper, so deep was his emotion, he recalled that day in London, 55 years before, "when I rode with my great master to his first lecture," and how supreme the influence of that acquaintance with Lister had reigned over his whole life thereafter. With this over he wished them well.

During the year that followed, his health slowly deteriorated and he went out very little. Gradually he became bedfast and died shortly before 9 p.m. on December 26, 1933, in his 87th year. He was buried at Pictou, near the scene of his early work.

Tributes from persons in all walks of life were forthcoming when his death became known. Dr. Murray MacLaren, then Minister of Pensions and National Health said in part: "His refined mind and tastes led him to be associated with all that was good, all that was patriotic, all that was noble."

The minutes of the Board of Governors of Dalhousie University used these words: "Dr. Stewart will be remembered as the surgeon who, in the Province, by his skill, raised the practice of that profession to a high plane. When he was in active practice he was the most highly respected and the best loved man in this Province."

In his memory, the Provincial Medical Board of Nova Scotia founded and endowed the Dr. John Stewart Memorial Lecture, given each year at the Dalhousie University Medical Refresher Course in Halifax. It was a humble but fitting tribute to a man, who by his character, high ideals and great professional skill, raised and ennobled the medical profession in his native province and far and away beyond its boundaries.

ORIGINAL ARTICLES

EPIPLOIC GRANULOMA DUE TO FISHBONE SIMULATING CARCINOMA*

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THE MAJORITY of granulomata occurring as a reaction to foreign substances in tissues are relatively easily diagnosed. The history of exposure to the irritating agent can usually be elicited, as in the case of paraffinoma of the face, breast or pleura, of lipoid granuloma of the lungs after the use of oily nose drops, or of granuloma following bronchography or hysterosalpingography. Granuloma of the rectum which may follow the injection treatment of hemorrhoids, beryllium lesions occurring in the lungs after exposure, and the small granulomata in the peritoneal cavity due to the powder on surgical gloves are all familiar to the medical profession. The authors would like to draw attention, however, to a form of granulomatous reaction which may produce marked abdominal symptoms and signs and which may be mistaken for extensive malignant disease. We refer to the epiploic granulomata produced as a result of perforation of the gastrointestinal tract by ingested foreign bodies, the fishbone being the most common offender.

Several reports of epiploic granulomata appeared in the European literature during the 19th century, but the first extensive account was published by Faber[1] who reported four cases in detail in 1898. He showed also that fishbones are dissolved fairly rapidly in normal gastric juice, but fail to dissolve in the presence of reduced gastric acidity. In 1907 Moynihan[2] drew attention to the difficulty of distinguishing between inflammatory tumours and carcinoma at operation. In 1927 Ginzburg and Beller[3] reported 12 cases of which five were due to chicken bones, one to a tooth pick and six to fishbones. They pointed out that in certain cases of obvious abscess, removal of the foreign body and drainage might be sufficient to effect a cure. Stetten[4] reported an additional case in the same year and Samet and Fogelson[5] reported another case in 1955. Feigen and Shapiro[6] in 1953 described a fishbone granuloma in a recto-sigmoid anastomosis two years after an anterior resection and emphasized the possible confusion with recurrence of the pre-existing neoplasm. The majority of published reports on this subject have come from continental Europe.[1, 7-17] The literature in the English language is surprisingly sparse. The nine cases due to fishbone mentioned above, appear to constitute the total number to be found in the English literature. Assorted etiological objects have been reported by other authors.[18, 19]

Occasionally a mass of localized fibrosis may be found in the omentum and may appear to be idiopathic, in that no foreign body is to be found within the fibrous tumour. A possible explanation for this curious lesion has been offered by Schmieden[20] and by Lewinsohn,[21] namely that some time later the foreign body has found its way back into the lumen of the gastrointestinal tract and has been passed with the stool. Our Case 4 may constitute an example of this phenomenon.

DIAGNOSTIC FEATURES

From a review of some 70 reported cases and from our own experience, we are struck by the frequency with which the initial event of perforation by the foreign body cannot be identified from the history. Many patients do not recall any symptoms before the onset of gradually increasing pain, colic and loss of weight. Occasionally the onset can be defined by sudden abdominal pain, chills and fever, but this is exceptional in those patients in whom granulomata proceed to form. As a general rule the symptoms are increasing discomfort, obstructive crampy pains, anorexia, alterations in bowel habit, abdominal distension and progressive weight loss extending over weeks or months. The similarity of the symptoms to those of gastrointestinal can-

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cer increases the diagnostic difficulties. Some localized tenderness in relation to an irregular mass is the most common physical finding.

Laboratory studies are not particularly helpful. Mild anemia may be present. Occult blood in the stool is not usual. The erythrocyte sedimentation rate may be increased and the gastric acidity may be low.

Barium enema examination is the diagnostic aid which is most generally useful. The palpable tumour is often larger than the appearance on barium enema would suggest. Both ends of the narrowed area of bowel tend to become cone shaped,[22, 23] as compared to the sharper cut-off seen in those with carcinoma. The mucous membrane is usually intact and may be puckered in accordion fashion due to the longitudinal shrinkage of the encircling fibrotic tissue. di Rienzo and Mosca[24] have described a pharmacodynamic test in which the injection of pitressin is followed by frank activity of the muscularis mucosa which may be observed under the fluoroscope. Their paper contains good illustrations of the radiological appearance of the bowel in response to this test. A barium series is indicated if signs of obstruction are not unduly prominent.

Operative Findings

The tumour may vary in size from 2 cm. to 15 cm. It may involve the stomach, bowel, lesser or greater omentum, the mesentery or the abdominal wall. The tumour may appear to involve both the stomach and the transverse colon. In such cases the appearance is possibly due to migration of the foreign body from the stomach into the greater omentum overlying the colon.

The gross finding of an irregular whitish mass of firm to hard consistency may lead readily to an initial operative diagnosis of extensive and perhaps inoperable malignancy. Careful examination will usually reveal that there is no evidence of metastatic spread and that no intrinsic tumour of stomach or bowel is actually present. A frozen section study of a fragment of the tissue may be highly desirable and informative.

Pathology

Pathological examination may or may not reveal a necrotic abscess cavity containing the foreign body. Subacute inflammation in the form of round cell infiltration is often present. The most striking finding is the presence of solid scar tissue due to more or less mature fibrosis which may be really remarkable in its extent. Giant cells may or may not be present.

Treatment

Reports in the literature reveal a wide variety of surgical approaches to the problem of treatment. Frank abscess cavities have been opened and drained, the foreign body being recovered in several cases. Smaller tumours not involving viscera have been excised. Resections of viscera have been carried out as for malignant disease. Enteroenterostomies have been performed to circumvent obstructions.

The simplest treatment which will eradicate the lesion is clearly all that is necessary. In many cases a cleavage plane along the serosa of the affected viscus can be defined and the tumour can be excised without entering the stomach or bowel. Frozen sections should be carried out on larger tumours if there is the slightest suspicion that the tumour may be benign. Resection limited to badly involved bowel may be necessary on occasion. If the possibility of granuloma is kept in mind and the lesions are explored carefully, it should be possible to avoid unnecessarily radical operations.

Case Reports

Case 1.—A 47-year-old watchman was admitted to the Reddy Memorial Hospital, Montreal in May 1951. He gave a history of recent loss of appetite, constipation, distension and some crampy mid-abdominal pain. There was no loss of weight. He had been observed for six months because of a pulmonary lesion without proof of the diagnosis of tuberculosis. On examination he did not appear ill. Mild abdominal distension was noted and a large tumour mass was present in the mid-abdomen which was mildly tender. There were no other relevant physical findings.

Laboratory studies revealed a mild anemia, an increased erythrocyte sedimentation rate

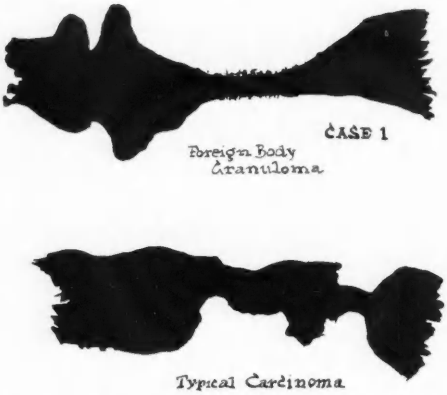


Fig. 1.—Case 1. Sketch of appearance of transverse colon on barium enema. The conical ends of the strictured bowel are shown, with the puckered but otherwise normal mucous membrane. The lower sketch shows the sharper cut-off and greater mucosal distortion of an extensive carcinoma in a similar location.

and reduced gastric acidity. The urinalysis was normal. Barium enema showed an area of stenosis in the transverse colon which was considered to be due to an extrinsic lesion. Unfortunately, the original films were subsequently destroyed. A sketch of the lesion with a comparative representation of a typical carcinoma of similar extent is illustrated in Fig. 1.

Operation was performed on May 22, 1951. There was no free fluid in the abdominal cavity and the liver was normal. The tumour was adherent to the abdominal wall from which it was separated with difficulty. The tumour measured 15 cm. in size and involved the entire omentum, most of the transverse colon and the greater curvature of the stomach. The surgeon (W. E. Kunstler) was convinced that the lesion was a carcinoma and frozen section was not performed. What was considered to be a palliative resection was carried out, with removal of 21 cm. of transverse colon and part of the greater curvature of the stomach together with the greater omentum. End-to-end anastomosis of the flexures of the colon was performed. The patient recovered satisfactorily and was discharged four weeks later. Six years later the patient was examined and was found to be well and working.

Pathological study of the specimen revealed a normal colonic mucosa with no evidence of carcinoma. In the centre of the hard, whitish-yellow tumour there were multiple abscesses, one of which contained a large fishbone. Microscopically, the mass consisted of fibrous tissue with diffuse subacute inflammatory and patchy granulomatous reaction. There was no evidence of tuberculosis or neoplasm (Figs. 2, 3, 4 and 5).

Comment

The extremely extensive epiploic granuloma misled the surgeon into the belief that malignancy was present. In this case the lesion engulfed the transverse colon to an extent which in all probability rendered resection necessary. Nevertheless, awareness of the possibilities inherent in the lesion, coupled with the use of frozen section, might have led to the correct diagnosis at the operating table.

Case 2.—A 69-year-old man was admitted to the Reddy Memorial Hospital in December 1951. For some two months he had experienced severe left upper abdominal and left shoulder pain immediately after a meal. He had given up eating solid food because of the pain which it caused, and had lost 35 lb. weight. Abdominal examination showed some left upper abdominal and left costovertebral tenderness but no mass. There were no pertinent physical findings except those due to recent weight loss. There was mild anemia; his urinalysis was normal. An intravenous pyelogram was normal. Barium series showed a definite filling defect on the greater curvature side of the fundus of the stomach highly suggestive of carcinoma. Again, a sketch is used to depict the appearance of the original radiograph (Fig. 6).

Laparotomy revealed a tumour about 8 cm. in size, which the operator (F. N. Gurd) considered at first to be a carcinoma of the stomach at the site shown by the radiograph. However, close inspection showed that the mass involved the upturned end of the greater omentum which was firmly adherent to the fundus of the stomach, and it proved to be possible to dissect the tumour away from the stomach and from the gastrosplenic ligament. The stomach was seen then to be apparently normal. The omental mass was removed by resection of the omentum. Further search was made for a primary carcinoma as it was presumed that the tumour was a large secondary lesion. However, no other disease was found and the abdomen was closed.

Pathological study revealed a fishbone in the centre of the tumour. Microscopic findings were similar to those of the previous case (Figs. 7, 8). Postoperatively, the patient

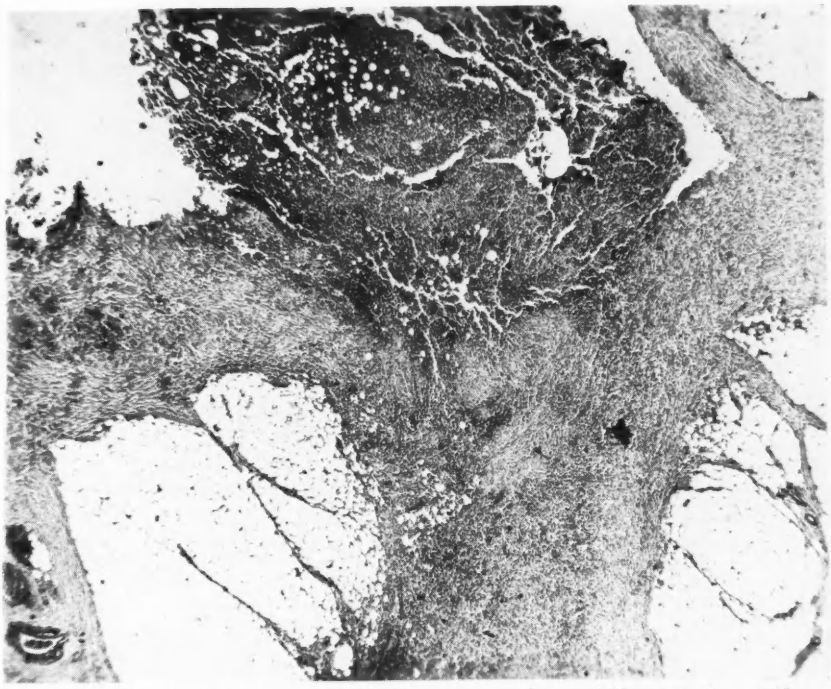


Fig. 2.—Case 1. Low power view of inflammatory exudate and fibrosis in the neighbourhood of the fishbone in omental fat (original magnification x 25).

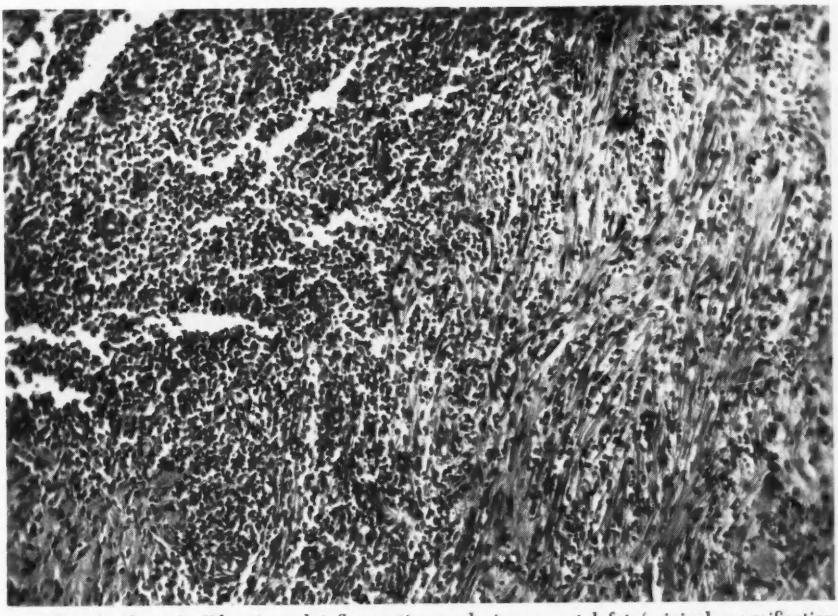


Fig. 3.—Case 1. Fibrosis and inflammation replacing omental fat (original magnification x 100).

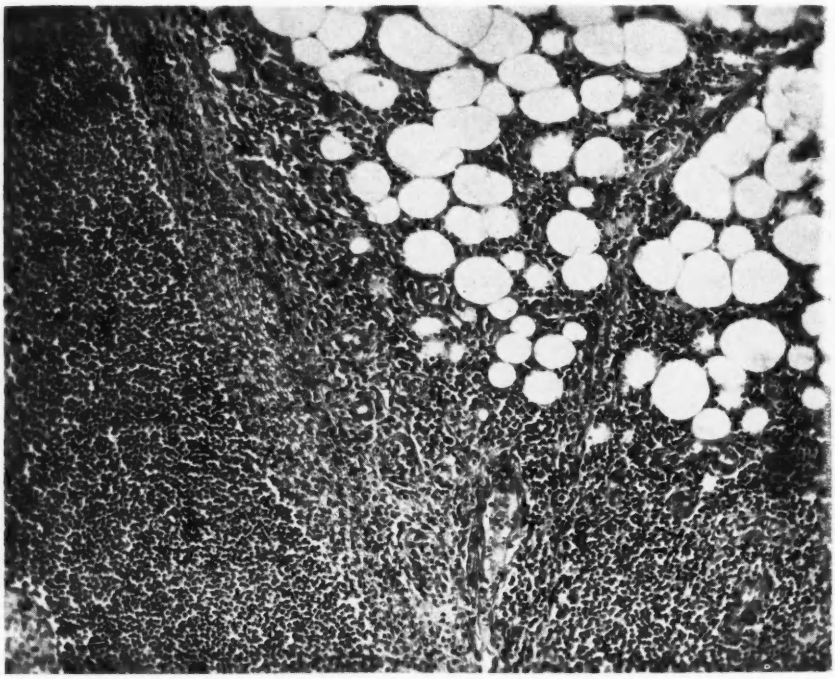


Fig 4.—Fibrosis and inflammation replacing omental fat (original magnification x 100).

added a further note to the history, conceding that his favourite breakfast was young trout, which he ate as one would eat a banana, washed down with beer.

The operation relieved his symptoms completely, and follow-up examination six years later revealed that he had remained in excellent health.

Comment

This case is one of straightforward perforation of the stomach with capture of the fishbone by the greater omentum. Symptoms were severe and the patient's life was in jeopardy due to inanition. Complete cure followed conservative operation, despite the fact that the correct diagnosis was not suspected at time of operation.

Case 3.—A 52-year-old man was operated upon by one of us (D. W. Ruddick) in the United Kingdom in 1949. The patient gave a six-week history of pain in the left lower quadrant, with a two-week period of alternating constipation and diarrhea. Some years before he had undergone subtotal gastrectomy for peptic ulcer. On examination he was seen to be poorly nourished. The abdomen revealed a hard, irregular mass in the left lower quadrant. Barium enema demonstrated an obstructing lesion of the left transverse colon with mucosal ulceration. No other significant findings were evident except for moderate anemia.

At operation a dense, hard mass was found which appeared to arise from the transverse colon. In mobilizing the mass several small abscesses were entered. The transverse colon was mobilized and resection was carried out by the Mikulicz method.

The specimen consisted of a portion of transverse colon surrounded by a dense mass of tissue causing partial obstruction. Mucosal ulceration was present. Within the mass was an abscess cavity containing a fishbone. Microscopic study showed a similar picture to that seen in the first two cases, namely inflammation, fibrosis and no evidence of malignancy. The patient eventually recovered.

Comment

Once again the predilection of fishbones for the region of the transverse colon is demonstrated by this case. The patient

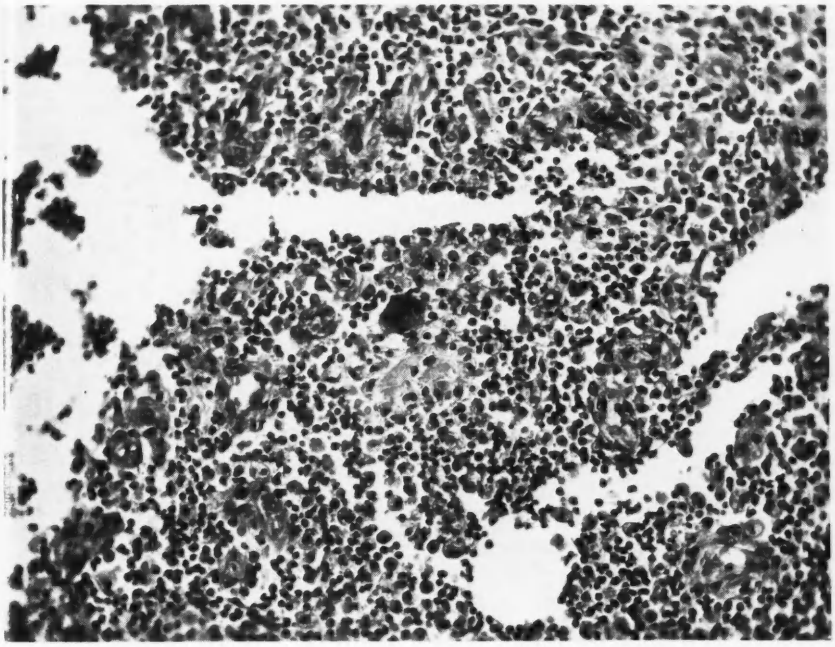


Fig. 5.—Case 1. High power view showing granulomatous reaction and giant cells (original magnification x 200).

presumably had a decreased gastric acidity as a result of his previous gastrectomy. Obstruction of the colon was almost complete and mucosal ulceration was present. The latter finding is unusual.

Case 4.—A 29-year-old mother of two children was admitted to the Montreal General Hospital on August 31, 1960. Her past history had been characterized by frequent respiratory infections. Otherwise there were no contributory findings in her past history. Two days before admission to hospital she had noted a dull ache in the left lower abdomen. There were occasional lower abdominal cramps and some flatus, no nausea or vomiting and no distension. Her last menstrual period, 10 days before, had been normal. There were no urinary symptoms.

On examination her temperature was 99.4° F. and her pulse rate was 88 per minute. The patient appeared well. Abdominal examination revealed a fixed mass about 7 cm. in diameter below and to the left of the umbilicus. The mass was mildly tender. Rectal examination was negative, as was pelvic examination. There were no positive findings.

Urinalysis was normal, her hemoglobin was 11.7 g. %, her white blood count 10,500/c.mm. and her sedimentation rate was 12 mm./hour. A flat film of the abdomen was normal. Barium enema showed distortion and indentation of the distal transverse colon, particularly upon compression of the mass. The mucosal pattern was normal.

No definite preoperative diagnosis was possible. Laparotomy was performed (F. N. Gurd) on September 3, 1960, under general anesthesia. There was no free fluid in the abdominal cavity and no intra-abdominal abnormality other than the tumour mass. The mass was adherent to the anterior abdominal wall but was dissected free. It measured 7 cm. in diameter and was about 2 cm. to 3 cm. thick, resembling a hamburger in size and shape. It was mottled whitish-grey in colour, and was embedded in omentum and firmly adherent to the anterior surface of the transverse colon. It was possible to excise the left half of the omentum together with the mass. A cleavage plane was found between tumour and colon, revealing no disease in the bowel itself except for some thickening of the apposed bowel wall. No evidence of perforation, recent or remote, could be demonstrated. A frozen section showed no

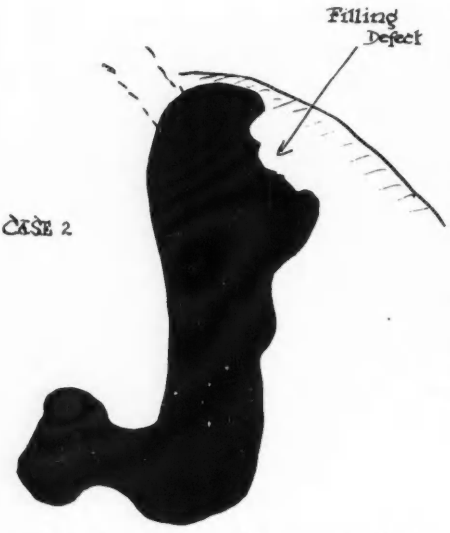


Fig. 6.—Case 2. Sketch of barium series showing indentation of gastric fundus: resemblance of filling defect to carcinoma is evident.

evidence of malignant disease. The appendix, which was normal, was removed and the abdomen was closed.

The pathology report read "localized fibrosis with mild non-specific inflammatory reaction in omentum. The fibrosis varies from young and highly active to mature, dense, collagenous tissue and hyalin. There is patchy hemorrhage, fibrinoid edema and surface fibrinous exudate. In some masses of fibrin there are large numbers of proliferating mesothelial cells. The lesion is not neoplastic; the etiology is not apparent. It is possibly related to the entity of retroperitoneal and mediastinal fibrosis." No foreign body was found within the mass, nor were there any giant cells.

The patient was discharged on September 12, 1960, and has since remained well.

Comment

The case remains unexplained and of course cannot be considered to be one of a proven foreign body granuloma. Nevertheless it is included here as an example of a lesion which could well have been mistaken for malignant disease and sub-



Fig. 7.—Case 2. Low power view of cavity in omentum which contained the fishbone (original magnification x 20).

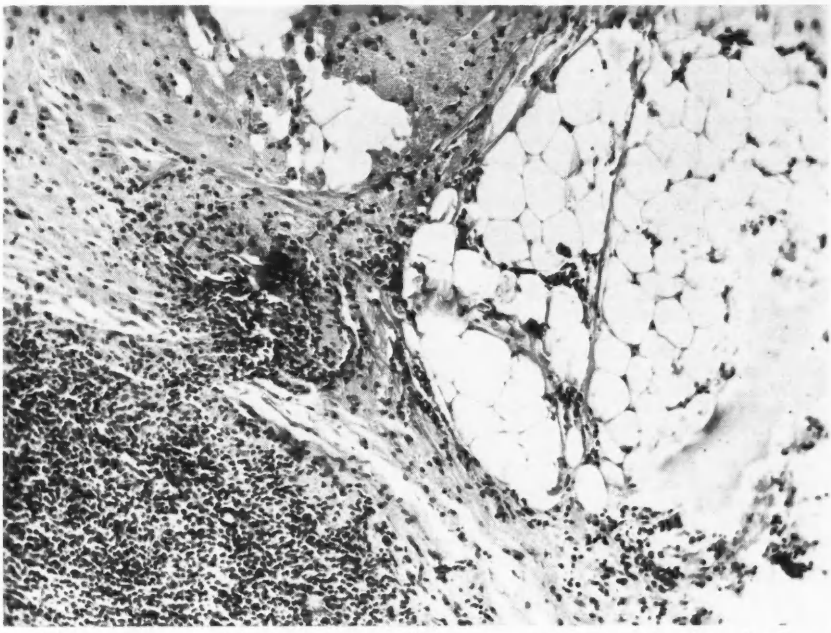


Fig. 8.—Case 2. Higher power view showing characteristic combination of fibrosis, inflammatory exudate and omental fat (original magnification x 100).

jected to radical surgery. It is not impossible that a foreign body had re-entered the bowel at some earlier date, as has been suggested by other authors.[20, 21]

Summary

A review of foreign body granulomata reported in relation to the gastrointestinal tract has been presented.

The diagnostic features, operative findings, pathology and treatment have been discussed. The diagnostic importance of radiological studies has been emphasized. The prognosis is generally good when the bulk of the tumour is removed and intestinal obstruction is relieved.

Three proven cases of epiploic granuloma due to fishbone are reported, all of which simulated carcinoma.

A case of a fibrous tumour in which no foreign body was found is also reported because of the diagnostic difficulties it presented. The possibility is considered that a foreign body may have re-entered the bowel in this case.

Acknowledgments

The assistance of Dr. Jean-Pierre Jean, Radiologist, and Dr M. B. Mackenzie, Pathologist, The Reddy Memorial Hospital, Montreal, is gratefully acknowledged. Thanks are also due to Dr. Weste and Dr. Goldberg for help in compiling the bibliography.

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RÉSUMÉ

Le granulome de l'épiploon peut atteindre tous les viscères de la cavité abdominale, y compris le péritoine pariétal et la paroi abdominale. Un certain nombre de ces granulomes sont dûs au passage de corps étrangers à travers l'estomac ou les gros intestins. Parmi ces corps étrangers, les arêtes de poisson en sont le plus souvent la cause. Entre 70 et 80 cas ont été décrits, dont seulement neuf ont été observés en Amérique du Nord.

Le diagnostic différentiel de cette entité est d'autant plus important qu'il est facile de le confondre avec un cancer abdominal ayant abouti à une occlusion intestinale qui nécessite une intervention chirurgicale d'urgence. Dans ces conditions, il arrive que les lésions impressionnantes font supposer au chirurgien un état si avancé de la maladie cancéreuse qu'il abandonne toute intervention même palliative. Seulement un examen histopathologique effectué au moment de l'opération peut révéler la nature des lésions. Il est vrai que dans certains cas, l'examen par les rayons X ou des réactions pharmacodynamiques puissent la suggérer.

Le pronostic du granulome d'épiploon est favorable à condition que les masses de tumeur qui causent l'occlusion ou la perforation intestinal aient été enlevées. Des cas de guérison spontanée ont été décrits.

Trois cas de ces granulomes ont été observés à l'Hôpital "Reddy Memorial" de Montréal. Ils étaient dûs à l'ingestion d'arêtes de poisson.

POSTMORTEM HOMOGRAFTS. James Barrett Brown, M.D., F.A.C.S. and Minot P. Fryer, M.D., F.A.C.S., Plastic Surgery Service, Department of Surgery, Washington University School of Medicine, St. Louis, Missouri. 64 pp. Illust., Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.00.

This book describes the use of postmortem skingrafts which in many instances have saved the lives of patients with extensive third degree burns. The difficulties of gaining consent for removal of this tissue for homologous transplantation are described and the authors have overcome this problem by appealing to the humanitarian feelings of the relatives in a sympathetic manner.

The indications for use of postmortem skingrafts and methods of application are described. The equipment for operation of a Skin Bank in a hospital is described, and laboratory experiments to support the usefulness of this method of coverage of extensive granulating wounds is presented. This book is recommended for plastic surgeons, general surgeons and for those in accident services.

SURGICAL MANAGEMENT OF RECURRENT CARCINOMA OF THE CERVIX

H. H. ALLEN, M.D., F.R.C.S.[C],* *London, Ont.*

THERE IS still much controversy about the place of surgery in the treatment of carcinoma of the cervix. In many centres it is now believed that radiation plus radical surgery should be used in all cases where the disease is confined to the pelvis and is surgically resectable. Some feel that the improved survival rate is sufficient to justify the increase in complications and morbidity with this method of treatment.

Radiation is still the primary method of treatment at the Ontario Cancer Foundation London Clinic. However, some indications for operation[9-11] are listed in Table I. It is in the last of these (poor response to radiation) that the author has been particularly interested over the past four years.

The radiation dosage used in the Ontario Cancer Foundation, London Clinic, is as high as that employed in most centres. The treatment consists of intrauterine and vaginal radium insertion followed by cobalt 60 therapy to the parametria. This treatment provides a dosage of approximately 8500 rads to point A and 5500 rads to point B.

Radiation Treatment of Recurrence

During the period between 1949 and 1954 there was no surgical treatment of recurrent carcinoma of the cervix at the London Clinic, re-irradiation being the only form of therapy employed. During this period there were 96 cases of recurrent carcinoma of the cervix, approximately one-half of them recurring within six months of treatment and about 15% recurring after two years. The sites of recurrence are shown in Table II.

This reveals that a rather high percentage of recurrences when first diagnosed, involved the cervix, vagina and parametrial tissue, which would theoretically have made them surgically resectable lesions. The symptoms at the time recurrence was first diagnosed are shown in Table III.

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TABLE I.—Indications for Operation

1. Carcinoma *in situ*
2. Young patient (may preserve ovary)
3. Pregnancy (1st and 3rd trimester)
4. Extension to bowel or bladder
5. Satisfactory radiotherapy not possible
6. Endocervical tumour
7. Intrauterine extension
8. Associated pelvic tumours
9. Pelvic inflammatory disease
10. Poor response to radiation

TABLE II.—Sites of Recurrence in 96 Cases

	Number of cases
Cervix and/or vagina	35
Parametrium	77
Pelvic wall	31
Extrapelvic	13
Unknown	2

TABLE III.—Symptoms Present at Time of First Recognized Recurrence in 96 Cases (1949 - 1954 Series)

	Number of cases
Vaginal bleeding or discharge	22
Low back pain	15
Leg pain and edema	20
Lower abdominal pain	31
Urinary	29
Bowel	21
Miscellaneous	7

TABLE IV.—Survival Time after Diagnosis of Recurrence in 96 Patients Treated by Re-Irradiation (1949 - 1954)

Months of survival	*Number of cases*
0 - 6	36
6 - 12	33
12 - 24	23
Over 24	4

These patients very frequently had pelvic discomfort, either in the lower abdomen or low back, but only about one-quarter of them had vaginal bleeding or discharge.

The outlook for these patients treated by re-irradiation for carcinoma recurrence is shown in Table IV. About one-quarter survived beyond 12 months and only four survived two years or longer. It should be pointed out, however, that none of these patients can be considered to have received radical re-irradiation. Of the 96 patients,

three were believed to have remained in Stage I and 34 had not advanced beyond Stage II when recurrence was first diagnosed. Thus it was thought that nearly 40% might have been salvable by some surgical procedure. Among the first to focus attention on the surgical treatment of recurrent carcinoma of the cervix were Heyman and Belonoschkin[6] who, in 1949, reported a five-year survival rate of 29.1% in recurrent carcinoma and 12.1% in residual carcinoma of the cervix.

Surgical Treatment of Recurrence

If the diagnosis of recurrence can be made before the disease has progressed to the point at which it is necessary to remove other pelvic organs, the survival rate is markedly improved.[8, 9, 12] Early diagnosis therefore is extremely important.

Diagnosis

Frequent visits to a follow-up clinic and awareness of recurrence upon eliciting any of the complaints outlined in Table III are most important. They are especially important for the patient who has been symptom free, even if only for a short period of time, and then begins to complain. Careful abdominal and pelvic examination, the latter always including rectovaginal palpation, should be carried out at each clinic visit. Nodularity, ulceration, pelvic masses or asymmetry of the vaginal vault not previously noted, should certainly arouse suspicion.[1-4] All these points should be carefully recorded on the patient's chart at each clinic visit to provide a base line. Occasionally, hydronephrosis alone may be the first evidence of recurrent disease although this may occur as a complication of radiation treatment.[3, 4] In our experience it most often indicates recurrence. Graham reports[5] that vaginal smears will be positive in about half the cases before the diagnosis can be made on physical examination. Vaginal smears at each follow-up visit are therefore desirable.

The patient suspected of having a recurrence may lose valuable time waiting for biopsies to become positive or for definite enlargement of pelvic masses.[3-12] Absolute diagnosis may be difficult, but examination under anesthesia, and if necessary, laparotomy to be certain of the diagnosis, is not too radical in view of the extremely serious nature of the disease.[3, 4]

Surgical Cases

A study was also carried out on a group of 37 patients with recurrent carcinoma of the cervix, who had been operated upon between 1955 and 1959 with the objective of permanent cure of their disease.

Table V indicates the stage of their disease when it was first diagnosed and the stage of the disease when recurrence was recognized.

In almost every case the recurrent carcinoma was considered to be in a more advanced stage than at the time of primary treatment. It is felt that this situation could have been avoided by more intensive follow-up clinic care, and by more prompt action when recurrence was first suspected.

Preoperative preparation

Once recurrence has been diagnosed or seriously suspected, the patient should be subjected to careful physical examination with emphasis on assessment of her general condition and cardiovascular status. Attempts should be made to establish the diagnosis of recurrence by positive biopsies, whenever possible. Complete blood counts, hematocrit, non-protein nitrogen, plasma protein values and blood volume should be obtained. Radiographs of the chest and pelvic bones should be taken in an attempt to rule out possible distant metastases. Intravenous pyelograms or retrograde pyelograms are often extremely useful. If urinary obstruction is present it should be relieved and the patient's electrolyte and

TABLE V.—Stage of Disease at Time of Primary Treatment and at Time of Recurrence (1955 - 1959) 37 Cases

Stage	*At primary treatment* — *Number of cases*	*At recurrence* — *Number of cases*
I	8	1
II	22	8
III	6	8
IV	1	20

TABLE VI.—Type of Operations Employed in 37 Cases

Type of operation	Positive lymph nodes	Negative lymph nodes	Alive and well	Total
Hysterectomy and nodes	4	13	8	17
Exenteration and nodes	8	5	3	13
Inoperable	7	0	0	7
Total	19	18	11	37

other biochemical disturbances should be corrected insofar as possible before any major surgical procedure is undertaken. Cystoscopic and sigmoidoscopic examinations are generally indicated as well. Preparation of the bowel with either neomycin or a sulphonamide and streptomycin is advisable, as it is rarely possible to anticipate those surgical procedures on the bowel which may be necessary at the time of operation. Three to four thousand cubic centimetres of blood should be available for each patient.

Operative technique

Under general anesthesia the patient is examined again and unless there are new findings which contraindicate proceeding with the operation, laparotomy is carried out. At this time the peritoneal cavity, omentum, liver, bowel and periaortic glands are inspected. If no distant metastases are found, the periaortic glands are removed and a frozen section of this tissue is examined. If any tumour tissue is found in the periaortic glands we are very reluctant to proceed with the operation. If the frozen section of these glands does not reveal evidence of malignancy, and if the local mass in the pelvis seems resectable, we proceed with the operation.

The type of operation and the organs removed depends on the extent and nature of the disease. Frozen section from suspicious areas will very frequently be of some help in deciding how extensive the operative procedure should be. One should be prepared to be as radical as is necessary to remove the local disease, and the patient's chance of long-term survival should not be jeopardized by attempts to minimize the extent of the operation.

The problem of adequate and accurate blood replacement during operation is important. There are various methods by which the extent of blood loss may be estimated. One such method involves serial determinations of blood volume by radioactive isotope techniques. In the author's opinion a more accurate assessment of total blood loss is provided by the weighing of all sponges used throughout the operation.

The types of operation performed on this series of patients are recorded in Table VI.

Extension of the disease to the pelvic wall and involvement of the iliac vessels is not necessarily a contraindication to proceeding with the operation if there is no evidence of spread beyond the pelvis and if all tumour-bearing tissue, including the involved vessels can be excised. In two such cases in the series reported here, it was necessary to remove the internal and external iliac arteries and veins and part of the common iliac artery with insertion of a nylon arterial graft from the common iliac artery to the femoral artery. One patient survived seven months and died of bowel obstruction due to intraperitoneal extension of her disease. The second patient survived two years and although an autopsy could not be obtained it was believed that she died of an ascending urinary infection. Our chief experience has been with the procedure of anterior exenteration. One total exenteration with formation of a sigmoid bladder and proximal colostomy was carried out. After a short trial with the ileal bladder we have adopted the procedure of rectal bladder formation combined with a proximal colostomy so that the fecal stream does not cross the ureteral openings. These patients generally remain free of infection and quickly gain urinary control so that they void every two to four hours during the day, and with controlled fluid intake in the evenings get up to void only once or twice during the night. It is desirable that they void reasonably frequently to avoid pooling of urine and

possible hyperchloremic acidosis. In general they do better if they take a daily maintenance dose of sodium bicarbonate.

At the termination of the operation, the vaginal cuff is left open and a latex rubber drain with multiple openings, in either side of the pelvis, is attached to a Stedman pump. In this way the exact amount of pelvic drainage over the next 10 days to two weeks can be accurately estimated. This drainage is essentially serum with a small amount of blood and represents an appreciable loss of protein which must be replaced in the form of blood or plasma. This pelvic drainage is rather copious in the first 24 to 48 hours and gradually falls off thereafter, amounting to an average of about 300 c.c. per day for the first week to 10 days. In respect to their protein loss these patients are treated essentially the same as patients with burns and their protein requirements are essentially those of a patient with a 15% to 18% burn.

Postoperative care

The problem of protein replacement and electrolyte balance is extremely important during postoperative convalescence. Serum protein and electrolyte estimations are of help but in the author's opinion, a more accurate estimation of the patient's requirements is obtained by careful measurement of the body's losses during this period. This involves accurate estimates of the pelvic drainage and of the amount and composition of the urine. These, combined with the loss from the Wangensteen suction and the estimated insensible loss, provide a fairly accurate estimate of what should be replaced. Blood and plasma are used frequently to replace protein loss, and electrolytes are replaced as indicated. About the fifth or sixth day when the bowel begins to function, the Wangensteen suction can be discontinued and oral feedings slowly begun. A high protein, high vitamin diet is desirable as soon as it can be tolerated.

Complications

The complications encountered in this series of patients are listed in Table VII. Cardiac arrest accounted for the only operative death. One patient developed an embolism from the ligated end of the ovarian vein, eight months postoperatively. At autopsy no evidence of malignancy was found. Four fistulas occurred, two vesicovaginal, one ureterovaginal and one in a patient who had had a total exenteration in which the sigmoid colon was moved down and sutured to the anus and a sigmoid bladder formed in this manner. Infection in the postoperative period was easily controlled by pelvic drainage. Kidney failure occurred in one case in which bilateral marked hydronephrosis was present before the operation. The patient withstood the procedure quite well, made a very slow postoperative recovery and died in hospital three months later. The only pathological findings at autopsy were those of bilateral pyelonephritis.

TABLE VII.—Surgical Complications (1955 - 1959)

Cardiac arrest	
Embolism	
Fistula	
Infection	

Conclusions

Constant alertness in follow-up clinics is essential for early recognition of recurrence of carcinoma of the cervix.

From a study of 133 cases of recurrent carcinoma of the cervix it is concluded that patients with certain symptoms should be suspected of having recurrence and should be watched closely.

The survival rate of these patients may be increased by earlier use of laparotomy when suspicious symptoms and signs develop.

Valuable time is frequently lost by waiting to be absolutely certain that recurrent carcinoma is present.

Re-irradiation as carried out in this series of patients was inadequate. A surgical attack on pelvic recurrence appears to have some merit.

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Résumé

Le traitement du cancer du col utérin et ses récidives est encore sujet à discussion. Le présent article traite ce sujet, basé sur un ensemble de 133 cas de récidives vus et soignés au "Ontario Cancer Foundation, London Clinic, Ontario".

Pendant la période de 1949 à 1954, la thérapeutique par irradiation fut la seule utilisée dans les récidives: ceci concerne 96 cas; parmi ceux-ci, environ un quart fut prolongé au delà de 12 mois et quatre seulement survécurent plus de deux ans.

Trente-sept patients furent revus et traités chirurgicalement. A ce propos, l'auteur insiste sur la nécessité de diagnostiquer la récidive de façon aussi précoce que possible: ceci ne peut être fait que par une surveillance clinique très approfondie, poursuivie longtemps après la première intervention. Les symptômes de récurrence sont souvent assez vagues; il ne faut pas attendre des biopsies positive ou l'apparition de tuméfactions intra-abdominales pour se décider; il faut au contraire ne pas hésiter à faire des examens sous anesthésie, ces examens pouvant être suivis et complétés par une laparotomie exploratrice en cas de doute. Si la récidive est confirmée, il faudra se livrer à une résection chirurgicale très étendue, dont l'importance variera évidemment avec la nature des lésions rencontrées. Au cours d'une intervention de ce genre, la vérification des régions douteuses par l'examen histologique sur coupes à la congélation sera des plus utiles. En outre, il convient de s'entourer de toutes les précautions usuelles en grande chirurgie, en ce qui concerne la préparation du malade et les soins post-opératoires.

Dans ces conditions, et sans radiothérapie, des résultats encourageants ont été obtenus.

FACTS OF LIVING: PUZZLE OF COURTESY*

"Because the distinguished clinician who is called upon feels honored, not to mention deeply concerned and fraternally kindly, he customarily exercises his right to decline any fee; professional courtesy is one of our very old and courtly, and benign institutions.

"The only disadvantage (as some have begun to complain) is that it deprives us of medical care. Oh, there is no hesitation about asking a colleague to manage one's coronary occlusion; the fellows rally around. But we have succeeded in educating patients to seek aid for minimal symptoms, to have a chest film routinely, and above all undergo preventive examination. We do not ask the same consideration for ourselves, or at least seem reluctant.

"There never seems to be a proper way of discharging the obligation. There are ingenious, friendly ways: wives, in conspiracy, can discover that the consultant was changing over to 16-mm.-motion picture equipment and hadn't yet bought all the appliances. Many a physician's physician is left wondering if he should open a liquor store, a camping supply shop, or a theatre ticket agency. And in 'routine' situations of preventive medicine and the like, this solution is singularly inappropriate.

"Some even cry, Abolish professional courtesy! Naturally the most 'modern' and 'practical' attitudes appear best (at the time), but it would be judicious to understand why 'this day and age' is different and, before giving it up, exactly what is so troublesome about an ancient and meaningful custom."

*Editorial: *Medical Tribune*, Nov. 14, 1960.

ACUTE APPENDICITIS PRESENTING AS SCROTAL SWELLING: REPORT OF TWO CASES

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"APPENDICITIS is the most common lesion requiring intra-abdominal surgery in childhood" (Gross). Although the etiology and specific pathology of the disease are the same in the child as in the adult, the criteria for diagnosis are different; the diagnosis is frequently more difficult to make, and the missed case runs a more rapid and lethal course.[1-8]

Appendicitis is rare during the first year of life, and is infrequently encountered during the second year; from then on it is quite common, particularly from the age of six to 12 years, according to Gross.[4]

Anorexia and vomiting are probably the most constant symptoms, together with abdominal pain. In the very young child the lack of abdominal guarding and spasm may create major difficulties in the assessment of pain and tenderness. The temperature and white blood count are often of very little help in making the diagnosis.

The presenting symptoms, especially in the young child, may at times be quite misleading, even in the presence of an admittedly suspicious history, as illustrated by the two patients reported on in this paper who presented with tender, non-reducible right scrotal swellings. It is of historical interest that during the American Civil War, appendectomy was performed on an 11-year-old drummer boy because of a scrotal mass thought to be an incarcerated hernia, which at operation was found to be a hernial sac containing a gangrenous appendix.

The following two cases illustrate an unusual pattern of presenting manifestations of this common disease.

CASE REPORTS

V.A., a 36-month-old white boy was brought to hospital with a history of periumbilical pain. Two days before admission the child experienced the onset of moderately severe periumbilical pain which had passed away in a few hours. There was no associated vomiting. Over the next two days he had only mild abdominal discomfort but was noted to be anorexic.

Physical examination at the time of admission revealed a rectal temperature of 100.6° F. and a regular pulse of 100 per minute. The remainder of the physical examination was essentially negative, except that the abdomen showed some increase in muscle tone, but was otherwise unremarkable and non-tender to palpation. Rectal examination was also non-contributory. The right side of the scrotum was red and swollen. The mother stated she had noted this scrotal swelling for the first time about six hours before admission. On palpation there was a tender mass in the right side of the scrotum which could not be reduced. The right spermatic cord appeared to be thickened and tender. The left half of the scrotum and its contents were within normal limits. Bowel sounds were decreased. The preoperative diagnosis was that of a strangulated right inguinal hernia.

At operation a horizontal incision measuring approximately 3.5 cm. in length was made in the lowermost abdominal fold above the region of the inguinal canal. A large, firm mass was found in the inguinal canal extending through the external inguinal ring into the scrotum. This was made up of a very thick-walled, inflamed and edematous hernial sac, extending down to the testis, together with the spermatic cord which was also thickened and edematous. The testis and epididymis were essentially normal in appearance and consistency. The sac was densely adherent to the cord structures. The hernial sac was dissected free from the cord structures up to the level of the internal abdominal ring. The sac was then opened and purulent fluid released. The sac was otherwise empty. Culture of this fluid revealed only a light growth of non-hemolytic streptococci. Digital examination through the neck of the sac revealed what appeared to be an edematous appendix. The sac was then ligated at its neck by means of an 00 chromic suture and the redundant portion was amputated.

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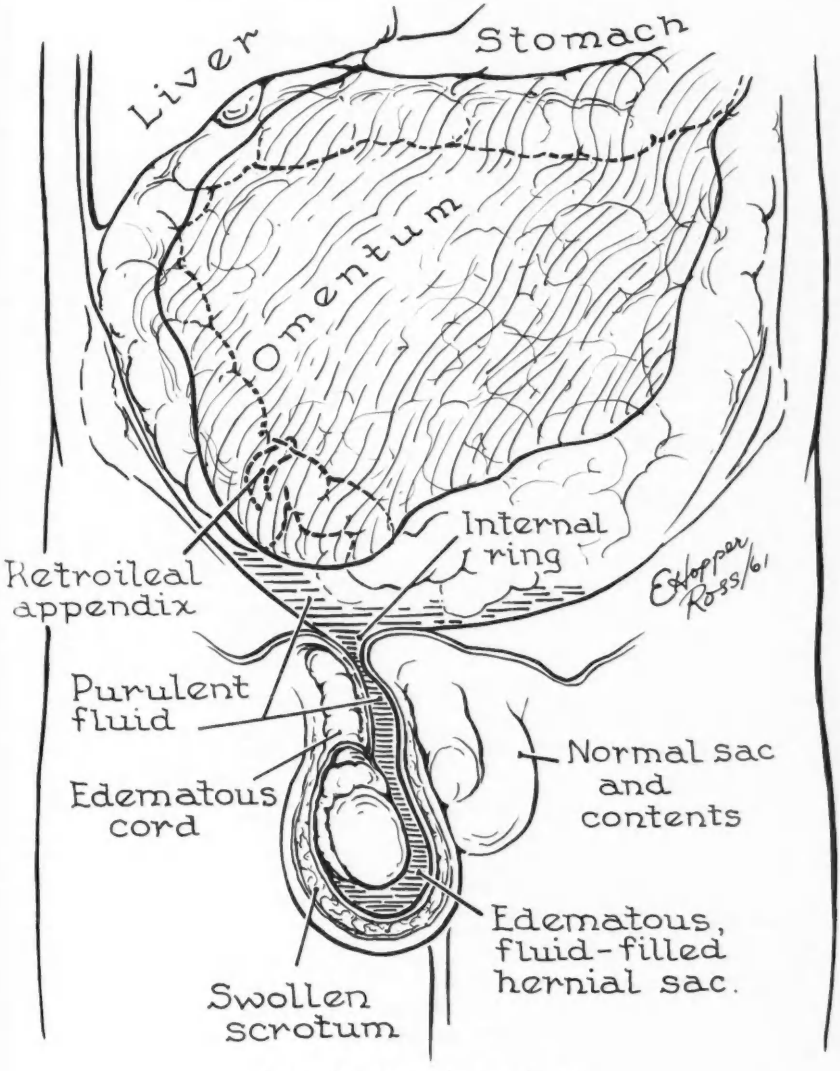


Fig. 1.—Operative findings in Case 1.

The internal ring admitted the index finger and was narrowed by means of two interrupted chromic 00 sutures. The testis was replaced in the scrotum and the usual layer closure was carried out. The abdomen was then redraped and a high McBurney incision was made. Upon opening the peritoneum purulent fluid similar to that found in the hernial sac, was encountered. A swab taken from this site was bacteriologically identical with the aforementioned specimen. The cecum was then mobilized and an acutely inflamed nonruptured appendix was found in the retroileal position (Fig. 1). There was a marked inflammatory reaction about the appendix and a moderate amount of shaggy fibrinopurulent exudate on the adjacent loops of small bowel. The appendix was amputated in the routine manner, and the appendiceal stump was inverted into the cecum by means of a purse-

string suture of 000 chromic catgut. The cecum and terminal ileum were then replaced into the abdominal cavity and a routine layer closure without drainage was performed. Interrupted 0000 black silk sutures were used to close both skin incisions.

Postoperatively the patient did well. He was maintained on intravenous fluids, and continuous gastric suction drainage until the second postoperative day and placed on pediatric Combiotic® 1 c.c. twice daily from the time of operation. This was discontinued on the sixth postoperative day, at which time he was discharged home. The incisions were well healed by primary intention. When seen in follow-up clinic one week postoperatively, there was only minimal residual non-tender scrotal swelling on the right side. He rapidly resumed normal activity and has remained well.

The pathological report was acute appendicitis with acute inflammation of the hernial sac.

Case 2.—C.D., a 21-month-old white boy was brought to hospital by his mother because of a swelling which she had noted in his right scrotum a few hours previously, and because he had been obviously unwell for the preceding four days. Four days before admission the patient had had a temperature of 102° F., accompanied by severe and protracted vomiting, "bringing up everything he ate". He had complained of what appeared to be mild abdominal discomfort. He was seen by his doctor and at the time, a diagnosis of tonsillitis was made, and penicillin therapy was started. He improved over the next two days but remained anorexic. On the day of admission he vomited once and had two loose bowel movements which were apparently followed by the appearance of the right scrotal swelling. He was generally lethargic and his mother thought that he seemed more ill than previously.

At the time of admission to hospital his rectal temperature was 103.4° F., and his pulse was 160 per minute. He was a pale, sick-looking child, restless and irritable, lying sometimes on his abdomen with his legs drawn up and sometimes flat on his back. Physical examination revealed moderate pharyngitis. Examination of the abdomen revealed that bowel sounds were decreased but not remarkably so. There was a sensation of fullness and questionable guarding in the right upper quadrant. Examination of both lower quadrants and of the rectum was essentially negative. Examination of the scrotum revealed an elliptical, slightly tender mass which could not be transilluminated nor reduced, and appeared to be arising from above the external inguinal ring. The testis could be palpated in the distal portion of the scrotum and felt normal. The left side of the scrotum was unremarkable. The child appeared mildly dehydrated and had not voided during the 12 hours before admission. His hemoglobin at this time was 12.[illegible] g. % though when repeated two days later after the child was well hydrated, it was only 9.9 g. %. The white blood count on admission was 6500 per c.mm.

The child was taken to the operating room and an inguinal incision similar to that used in Case 1 was made. Here again there was a bulging mass in the inguinal canal extending into the scrotum. Further dissection revealed a thick-walled edematous hyperemic hydrocele of the cord. This was dissected out intact. Aspiration of this hydrocele after it had been removed revealed that it was filled with a thin, custard-coloured, purulent material. A small hernia sac was also found proximal to the hydrocele. This was opened and was found to be empty, but a slightly fecal odour was noted and a small amount of seropurulent fluid escaped from the peritoneal cavity. The neck of the sac was suture ligated, the usual repair was performed and the inguinal incision was closed in the routine manner. Then the abdomen was redraped and a high McBurney incision was made in the right lower quadrant. Upon opening the peritoneal cavity more seropurulent fluid was found, this time with a definite fecal odour. The appendix was found in the right lateral gutter and beneath the liver in association with an incompete malrotation of the cecum (Fig. 2). A Weir extension was made in order to deal with the gangrenous appendix which was lying in a large pocket of purulent material in the lateral gutter just beneath the right lobe of the liver. The appendix was removed in the usual manner and the stump was ligated. As much purulent material as possible was aspirated from the peritoneal cavity; the incision was then closed in layers with a large corrugated rubber drain from the region of the appendiceal stump in the lateral gutter. This was brought out through the distal portion of the incision.

The child did well postoperatively. He was kept on gastric suction for two days and maintained on parenteral fluids until the third day, when clear fluids were started by mouth. He was placed on Crystamycin® 0.5 c.c. twice

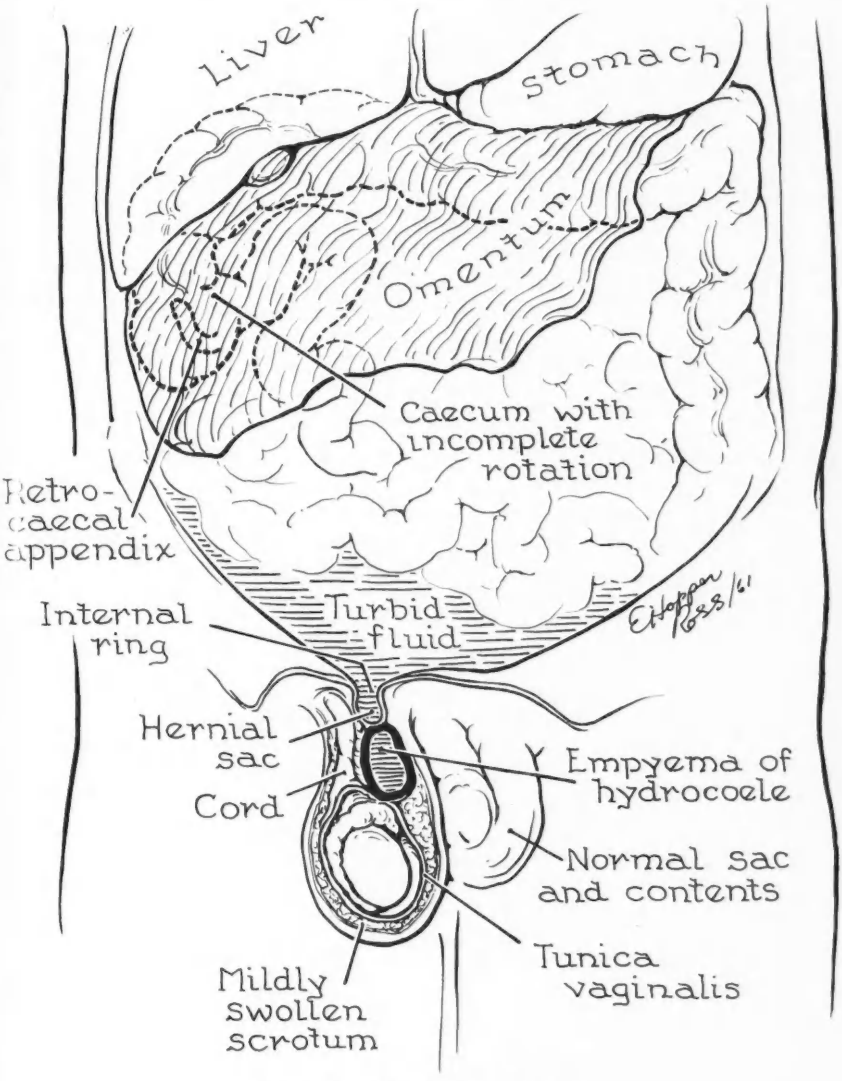


Fig. 2.—Operative findings in Case 2.

daily from the day of operation. There was a steady decrease in his temperature until it reached normal on the fourth postoperative day. The abdominal drain was removed on the sixth postoperative day and some drainage from the drain site persisted for the next three or four days.

The patient was discharged on the 12th postoperative day, and has done well since.

Cultures taken from the peritoneal cavity and from the fluid within the hydrocele both revealed *E. coli* and Bacteroides, resistant to streptomycin and neomycin but sensitive to the tetracyclines.

Both of these patients presented at the same hospital within a six-month period. These are the first two cases of appendicitis in the records of this hospital to have presented in this manner. Since both appendicitis and herniae are so common in boy children, one wonders why their coexistence is not encountered more frequently.

Summary

Two cases of appendicitis have been described, occurring in early childhood, an age at which this disease frequently poses major problems in diagnosis. The presenting manifestation in both cases was a tender, non-reducible right scrotal swelling. The details of their management have been discussed. Herniotomy and appendectomy were performed on both patients with a satisfactory outcome.

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Résumé

Deux cas d'appendicite survenus chez deux bambins à l'âge où cette condition peut poser de sérieux problèmes de diagnostic sont présentés. Dans les deux cas le point dominant de l'examen était la présence d'une tuméfaction irréductible et douloureuse scrotale droite.

Une herniotomie de même qu'une appendicectomie furent pratiquées par des incisions séparées chez les deux avec résultats excellents.

THE USE OF A DATUM*

"The ways of using a published article are many. Some among our colleagues belong to the razor-blade school and cherish the papers close to their hearts by excising them, abandoning without sorrow the shredded journal that is left. The wild ones insist they 'can find it when they need it,' meaning the select fragments, and of course W. C. Fields used to say the very thing as he clawed through his fabulous roll-top desk. The extremely sober students write for a reprint, sometimes get it, and preserve their journals and drawerfuls of reprints as well, for which, alas, there is needed an index.

"The library (when you can arrange to go there) does have several kinds of index, rather complete up to last February. The papers you have in mind were in May.

"Abstracts are an attempt to circumvent these troubles, and, as a superb form of note taking, accomplish a great deal for the mind that writes them. They are less than the data, but they can retrieve the data; now it is only a question of retrieving the abstracts. All that's wanted is an index. And maybe not, for each has a title, but the information is never looked for afterward under *that* title; it is always another reason that brings the matter up.

"These diverse forms of suffering come to mind because of new inventions by the chemists to make their information available. It must be splendid to work in chemistry and look up the data by clearly defined, fixed entities. By contrast, it is taxing to think in medicine, which is multilateral and endlessly evolving, even in its dimensions.

"If an idle genius can be found, the invention wanted in medicine is a manifold and infallible index, encyclopedic and pocketsized, that every physician can have in his desk drawer, and which guides him comprehensively to every datum he owns (or can expect to find in the library either) no matter how obliquely the occasion may evoke it. It is not, obviously, a list of chemical substances (it merely includes them) or a selection of nouns from the titles of papers (as if titles revealed much) . . . but only a genius can say what it is. The invitation is hereby extended—we will be obliged to him."

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*Editorial: *Medical Tribune*, Nov. 14, 1960.

SACROCOCCYGEAL TERATOMAS IN ADULTS

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TERATOMAS of the sacrococcygeal region are relatively uncommon; less than 100 authenticated cases have been reported.[5, 11, 22, 24] Recent reviews, however, have reported a large number of cases collected from the literature.[11, 20] The earliest record of a teratomatous malformation in the sacrococcygeal area was made in about 2000 B.C.[2] The first successful surgical excision was reported by Stanley.[26]

Sacrococcygeal teratomas are thought to be invariably present at birth, and about 90% of them are recognized at that time.[5, 11, 12] They appear only rarely in adults.[3, 11, 20] They are much more common in women.[5, 11, 22]

The rarity of these tumours in adults and the previously unreported occurrence in two sisters, together with successful surgical extirpation and a complete follow-up of four to six years, have prompted this report.

CASE 1.—L.S.L., a 28-year-old, married, well developed, white woman was admitted to hospital on April 24, 1954, complaining of low backache and a mass in the lower abdomen. She was unaware of this mass until she consulted her doctor because of sterility and inability to perform sexual intercourse satisfactorily. A large pelvic mass was discovered on this visit and hospitalization was advised. Menstruation had been regular and the last normal menstrual period had been on April 2, 1954. She had suffered from constipation ever since childhood and had taken laxatives regularly. She had no urinary complaints. There was no history of any congenital anomaly or of twins in the family.

Physical examination revealed a large, firm, fixed mass, the size of a 26 weeks' pregnant uterus in the pelvis, attached to deeper structures. No fetal parts or heart sounds could be made out. Rectal and pelvic examinations revealed a huge, cystic mass, situated posterior to the vagina and rectum. No cervix or uterus could be felt separately. Her blood pressure was 110/60 mm. Hg and her pulse rate was 90 per minute.

Investigations.—Her hemoglobin was 12.8 g. % (82%), with a total red blood cell count of 4.18 million/c.mm. and a colour index of 0.9. Her total white blood count was 6900/c.mm. with a differential count of neutrophils 74%, stab cells 4%, lymphocytes 21% and monocytes 1%. The platelets and red blood cells appeared normal. Her blood group was A. Wassermann and Kahn tests were negative. The urine and stool were normal.

Radiology.—Radiographs of the abdomen and pelvis showed deformity of the sacrum and absence of the coccyx possibly due to a congenital anomaly and associated with a spina bifida occulta of the sacrum. A large soft tissue shadow was visible in the pelvis. There was no evidence of calcification in this mass. The skull, the rest of the spine and the chest were normal. A barium enema showed the rectum to be displaced to the right side. The remainder of the colon was lengthened and markedly redundant.

Frog tests and Aschheim-Zondek tests of the urine were repeatedly positive.

Operation.—The patient was operated upon on April 28, 1954. A long, midline, infraumbilical incision was made. The uterus was small but normal in shape. Both tubes and ovaries were normal. There was an enormous cystic mass present behind the rectum and pelvic colon, pushing all the pelvic viscera forward against the abdominal wall. The pelvic peritoneum was divided between the uterus in front and the rectum behind, and the incision was carried laterally. Ureteric catheters were inserted preoperatively to prevent any damage to the ureters. Both ureters were clearly defined and the pelvic colon was mobilized. The cystic tumour could then be clearly visualized; 1000 c.c. of clear fluid was aspirated from the tumour and the cyst was laid widely open. It extended down to the sacrum, coccyx and perineum. On the anterior wall of the cyst at the level of the pelvic floor, a second semisolid tumour could be palpated. With careful dissection, the entire cyst and the semisolid tumour were removed completely. The semisolid tumour showed caseous

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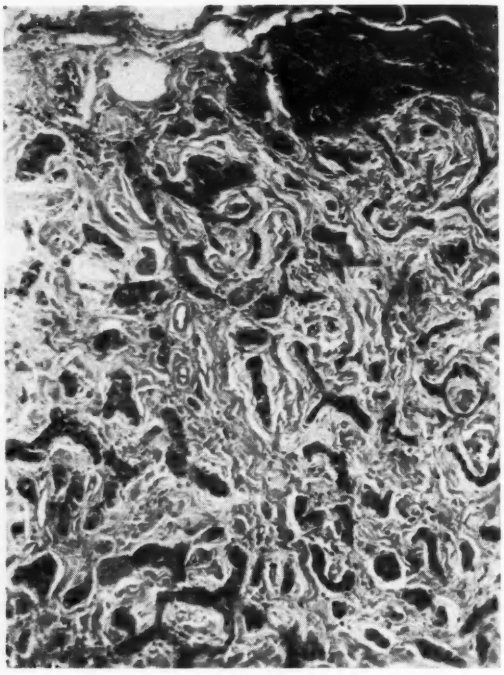


Fig. 1.—Wall of cyst containing an apparently invasive epithelial ingrowth which in none of 19 sections examined extended to the margins of resection. Endodermal components were lacking (x 100).

cheesy material. Most of the raw areas were reperitonealized. The wound was closed in layers with drainage. The ureteric catheters were removed at the completion of the operation.

The postoperative period was uneventful and the patient made an excellent recovery.

Histopathology.—Histopathological study of the excised tissue revealed an unusual congenital anomaly of the presacral region comprising a biloculated cystic structure with walls rich in smooth muscle. The smaller of the two locules was lined by stratified squamous epithelium and distended by keratotic debris and squames. Embedded in its walls were a few sweat glands and hairs.

The larger of the locules was bordered by a moderately heavy zone of acellular hyaline connective tissue so that with the smooth muscle bands in its walls, it had features of a lymphogenous cyst. In one area in the wall of this cyst, epithelium resembling that of sweat glands was growing as a highly differentiated carcinoma (Fig. 1).

The pathological diagnosis was a highly differentiated carcinoma (apparently arising from heterotopic sweat gland epithelium) in a presacral bidermal cystic congenital anomaly, growing in part as a so-called dermoid cyst.

The Canadian Tumour Registry, however, did not find evidence of malignancy in the cyst wall.

The patient was given postoperatively deep x-ray irradiation of 4000 r.

Follow-up.—The patient has remained symptom-free since operation and all the radiological investigations carried out recently have not revealed any local recurrence or distant metastases.

CASE 2.—R.L., a 28-year-old, married, well-built, white woman, younger sister of the previous patient, was admitted to hospital on July 9, 1956, complaining of a profuse, thin, yellowish discharge from the rectum for the previous month. She had no urinary complaints. Her menstrual history was normal. Date of the last normal menstrual period was July 2, 1956. She had delivered a normal full-term baby six months earlier but it had died immediately after birth.

Physical examination revealed a large cystic mass behind the rectum extending up to the anterior aspect of the sacrum. A pelvic examination was normal. Her blood pressure was 110/80 mm. Hg and her pulse rate was 80 per minute.

Investigations.—Her hemoglobin was 13.3 g. % (85%); total red blood cell count was 4.4 million/c.mm. and colour index 0.96. The total white blood count was 6200/c.mm. of which 59% were neutrophils. Platelets and red cells were normal; the mean corpuscular volume was 88 c.μ, and mean corpuscular hemoglobin concentration 34%. Wassermann and Kahn tests were negative. The urine was normal. Culture from the discharge grew colonies of coliform species, and rare colonies of paracolon species.

Radiology.—Radiologically, there was evidence of a congenital anomaly of the sacrum with marked deformity of the lower portion of the sacrum and a rudimentary or absent coccyx. On barium enema, there was displacement of the rectum and lower sigmoid to the right, and of the terminal ileal loops upwards, by a mass of soft tissue density which occupied the right half and the mid-portion of the pelvis.

Operation.—Operation was performed on July 18, 1956. A posterior "T" incision was made over the sacrococcygeal region and carried down to the bone. The coccyx was disarticulated, removed and the anococcygeal

raphe was divided. The rectum was pushed forward, entering the presacral space. The cyst was immediately opened and a large amount of thin, clear-coloured fluid escaped. Anterior to it was a second lesion which was secondarily infected and which had formed a fistula into the anal canal just inside the sphincter. Both the cysts were dissected out completely and removed. The fistulous communication into the rectum was closed. The wound was then lightly packed open. The upper and lower portions of the incision, however, were loosely sutured. Postoperative recovery was uneventful.

Histopathology.—On histopathological examination of the operative specimen it was noted that most of the cyst wall was lined by stratified squamous epithelium supported by inflammatory granulation and scar tissue enclosing strands of smooth muscle. Peripheral to this lay the fibro-fatty connective tissue containing occasional lymphoid follicles. At some points, the supporting collagenous connective tissues deep to the squamous epithelial lining contained microcystic structures lined for the most part by stratified, squamous epithelium, but with areas of a columnar type of epithelial lining (Fig. 2). Scattered gland-like structures were lined by a single columnar mucus-secreting type of epithelium. In some areas, the three varieties were intermixed.

The overall picture was consistent with an inflamed and probably infected sacrococcygeal teratoma.

Follow-up.—The patient has remained symptom-free and has no complaints.

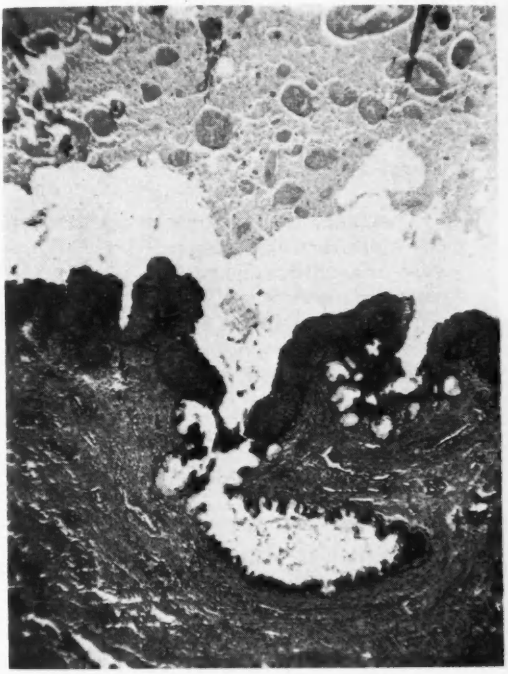


Fig. 2.—Wall of cyst lined by squamous epithelium, with gland-like structures and outpocketings lined entirely or in part by columnar mucus secreting epithelium (x 40).

Discussion and Comments

A review of the literature on the subject of sacrococcygeal teratomas has revealed a frequent interchange of the terminology of "dermoid cyst" and "teratoma". In most instances in which the term dermoid cyst was used, the tumour was a true teratoma.[7, 17, 29, 30] The attempt to classify sacrococcygeal growths as "dermoids", "teratoids", "included twins" and "fetal parasites" should be avoided; it is better to group all of these together under the one general term of "sacrococcygeal teratoma".[11]

As the teratomas combine the problems of congenital abnormality with those of neoplastic growth, it is small wonder that the literature concerning the origin and causes of teratomas is so confused. Similar benign testicular tumours occur in young horses.[28] Teratomas have also been produced experimentally.[17]

In our present state of ignorance only general assumptions as to the natural origin of these tumours can be made. Parthenogenetic development of the individual's primitive germ cells, migrating and lodging in the sacrococcygeal region might give rise to these tumours.[16] Other authorities believe that they arise primarily from cells already present in the sacrococcygeal region in the normal embryonic development, such as those of the postanal gut and neurenteric canal.[14] It can be said, however, that numerous theories still in acceptance should be discarded on the basis of recent advances in embryology and the excellent pathologic studies that have been recorded.[18]

Recent discoveries have made the family history and account of pregnancy, as with any other congenital abnormality, a matter of importance. The pregnancy may have been abnormal.[9] Although a significantly

higher occurrence of twinning has been reported in the history of these patients, other malformations have been rare.[11] Abnormal endocrine activity has also been reported.[23] The first patient described in this report also showed evidence of hormonal activity by repeatedly positive A-Z tests.

Teratomas are composite masses possessing more than one germinal layer. Wherever adequate examinations were made, tissues representing all of the three germinal layers were found in the large majority of cases. A wide range of tissue maturation may be found from case to case and indeed in different portions of one specimen. Some tumours are composed almost entirely of cells from a single germinal layer; careful search is required to demonstrate the other cell components and recognize the teratomatous nature of the mass. Sacrococcygeal teratomas may pursue one of two courses. They may persist as benign, well-encapsulated, cystic or solid lesions that grow at about the same rate as the host and are symptomatic only by virtue of their size or by displacement of adjacent organs, as in Case 1. Conversely, one element may at any time discard its mantle of benignancy and burst forth as a rapidly growing tumour. In a few instances, even incomplete removal seemed to stimulate an apparently innocuous lesion into hopeless malignancy.[11]

The growths present a wide variety of gross appearances. In general, largely cystic masses are more apt to be relatively benign, although such relationship may not always be found. Usually both solid and cystic forms are intermingled in an irregular way. Some specimens contain hair or sebaceous material; others have clear yellow or cloudy fluid. Microscopically, all types of tissues have been encountered.

These tumours may attain a large size before they are discovered, as was noted in our patients. They originate either dorsally or ventrally in relation to the sacrum.[9] The patient usually seeks medical care because of secondary symptoms which develop as a result of interference in function of the adjacent organs due to pressure by the growing mass.[8, 25] Such symptoms include disturbances in micturition, defecation, backache or sciatica. Inability to perform sexual intercourse and sterility were the presenting symptoms in our first patient. Infection with formation of a sinus may be another symptom, as in our second case. The large majority, however, complain of a mass. Increased development may be seen in some cases.

A tumour mass is visible in the majority. Associated congenital anomalies such as a three-lobed left lung,[16] a meningocele with spina bifida,[1] imperforate anus,[24] club foot,[4] and simple spina bifida,[21] have been reported.

The usual complications of these lesions are due to pressure, obstruction of the rectum and urethra with compression of the ureters against the pelvic brim, necrosis of skin and fistula or sinus formation. Infection may result from necrosis of the tumour and ulceration of the skin or rectum, or wound breakdown after incomplete removal. The incidence of malignancy has been reported as varying from 9%[5] to 17%[22] in the reviewed cases. Appearance of malignancy did not seem to be correlated with the duration of symptoms or with the size of the tumour.

In the differential diagnosis, meningocele[6] can sometimes be easily excluded by physical examination.

Neurological defects in the legs or radiological evidence of sacral deformity are highly suggestive of myelomeningocele. Myelography seems unnecessary, but in cases of doubt, it may be of help.

Chordoma may occur in this region in adults. It is more frequent in men and characteristically destroys the regional bone which is a rare finding in sacrococcygeal teratomas.

Exceedingly rare tumours, such as ependymomas, may also be present in the sacral area. These originate from ependymal cells lining the central canal of the cord and erode bone by pressure necrosis.

Pilonidal cysts and sinuses may be easily differentiated. In some cases, however, it may be impossible to differentiate a small teratoma with a draining sinus from an infected pilonidal sinus. Only histological studies will provide the correct diagnosis.

Diagnosis may be helped by the aid of

rectal examination, cystogram, barium enema and roentgenograms of the pelvis. Large numbers of cases may show calcium within the tumour on radiography.[22] The presence of bone or teeth within the lesion, or erosion of the neighbouring bony structures may be found. Aspiration biopsy is usually not advocated because of the inherent danger of dissemination.

Surgical removal is the treatment of choice regardless of the size of the tumour. If the tumour can be completely extirpated the prognosis is good. If skeletal or pulmonary metastases are present, obviously little will be accomplished by local excision. In the majority of instances the perineal or posterior approach has been used. The entire coccyx should always be excised along with the neoplasm, because microscopic nests of neoplastic cells are commonly found in, or immediately adjacent to this bone. In none of the cases reviewed, except those of Jones[15] and one of ours, has an anterior approach been utilized. A combined abdominal and sacral approach has been suggested if the tumour should prove too large or too extensive for a simple posterior excision.[4]

Preoperative irradiation is unwise because of the usual resistance of teratomas to irradiation[13] and because of the problems introduced by radiation of the pelvis, sterilization of the gonads and damage to epiphyses. In cases in which malignant change has been found in the excised tumour, as in our first patient, postoperative irradiation may be of value,[22] but it appears to be largely symptomatic and palliative. At present the only hope of cure appears to be early and adequate excision.

From the available statistics,[11] it is obvious that the outlook is far better for the sacrococcygeal teratomas in the newborn group than for those in older subjects. Pathologic examination of these neoplasms does not necessarily predict accurately the prognosis in any given case, as is illustrated by the report of our first patient. An optimistic attitude should be adopted towards sacrococcygeal teratomas, because now there is adequate proof that a good number of such patients can be cured if prompt and adequate surgical removal is undertaken.

Summary

A summary of the knowledge relating to teratomas in the sacrococcygeal region, commonly reported in the literature as "dermoid cysts", is presented.

Two successfully treated cases in two sisters, with a long term follow-up, are presented.

Acknowledgment

Our thanks are due to Dr. J. S. Campbell, Associate Professor of Pathology, University of Ottawa, for the histopathological study of the material; and to the Medical Superintendent, Ottawa General Hospital, for allowing us to publish these cases.

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Résumé

Cet article présente les cas de deux soeurs âgées d'environ 28 ans, présentant une pathologie très semblable. Dans les deux cas les symptômes étaient: présence d'une masse abdominale dure, située dans la région basse du ventre entre le rectum et la paroi abdominale postérieure, adhérente aux plans profonds; des douleurs vagues dans la région sacrée et une constipation chronique complétaient le tableau. Ces tumeurs furent enlevées, l'une par laparotomie et l'autre par voie perinéale; les suites post-opératoires furent très bonnes. Il s'agissait les deux fois de tératome sacro-coccygien.

Dans la littérature, une certaine confusion existe quant à la terminologie et la classification de ce type de tumeurs. On parle indifféremment de tératomes coccygiens de kystes dermoïdes ou de teratoïdes. Ces tumeurs combinent deux facteurs importants: une malformation congénitale et une croissance néoplasique. L'embryologie n'apporte que peu de lumière sur le premier de ces points: on sait que l'on y retrouve toujours des tissus dérivés des trois feuillets primitifs; des phénomènes de parthénogénèse ont été invoqués.

Le traitement de choix est l'ablation chirurgicale totale, sans considération de la taille; le coccyx devra être réséqué, car des petits ilôts cellulaires néoplasiques peuvent s'y trouver, provoquant des récidives. Lorsque l'ablation a été bien complete, le pronostic est bon.

DEMONSTRATIONS OF PHYSICAL SIGNS IN CLINICAL SURGERY. Hamilton Bailey, F.R.C.S., F.A.C.S., F.R.S.E. Emeritus Surgeon, Royal Northern Hospital, London, Consulting Surgeon, Italian Hospital, General Surgeon, Metropolitan Ear, Nose and Throat Hospital, Vice-President, International College of Surgeons. 13th edition. 928 pp. Illust. John Wright & Sons, Ltd., Bristol, England; The Macmillan Company of Canada Limited, 1960. $12.75.

Twenty editions, five in foreign languages, and 11 reprintings in 33 years should be ample recommendation for a book on any subject. This is the success story of Hamilton Bailey's 'Physical Signs in Clinical Surgery' which has now appeared in a completely new form encompassing an unique and fascinating collection of what must include all the known physical signs and clinical diagnostic methods available in surgery today. The illustrations are superb in quality and quantity, and in teaching value, and the reader is never required to hop about from page to page to place the written text with its visual accompaniment.

In this modern day of laboratory worship, it is encouraging to see just how far to reaching the correct diagnosis a careful physical examination can carry the surgeon. One's only regret is that its present size makes it more of a text than was its predecessor the coat-pocket companion. This classic, however, remains supreme in its field and must be required reading for every medical student. It will, no doubt, also remain the close ally of the fellowship candidate as well as a bedside consultant of the practicing surgeon and teacher.

LONGEVITY IN GASTRIC CANCER*

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OF ALL THE diseases that beset man, few are more insidious in their onset or pursue a more lethal course than carcinoma of the stomach. While the death rate from gastric carcinoma is sizable in most hospitals (2.5% of the last 12,000 postmortem examinations at the Vancouver General Hospital), statistics show that throughout the North American continent the rate is slowly but steadily declining. The reason for this is not clear. In British Columbia the drop was from 26.2 per 100,000 population in 1937 to 19.7 per 100,000 in 1957.

In a recent private canvass of surgical colleagues in Vancouver, none could recall from personal experience more than one or two instances in which a patient with gastric cancer had survived five years following operation, and most could recall no such instance. When such survivors were recalled, it was not uncommon for the surgeon to volunteer surprise that this was the case, in view of the extent of the disease found at operation which had led to the performance of a restricted resection with palliative intent only. For this and other reasons it was considered that a comparative study of all the features related to the neoplasm and its host in such a group of long-term survivors compared to those of a selected group of short-term survivors, might bring forth some significant features common to longevity or to rapid dissemination and early demise.

Thirty-six well documented cases of cancer of the stomach who survived five to 10 years after gastrectomy comprised the long-term survivor group. Some of these are still living with or without demonstrable disease; others have died of their malignancy or from some unrelated cause. The average period of survival was eight years. An equal number of cases of gastric cancer with survival of less than four years after gastrectomy were selected from local hospital records of the same period. The average duration of survival of these subjects was 19 months. Their selection was designed to provide for more realistic comparison of the two groups as shown in Fig. 1. Only those cases were included in which the objective of treatment had been definite cure by surgical ablation, and in which the findings at operation indicated that the disease was confined to the stomach and perigastric lymph nodes. If the growth was found to be adherent to surrounding structures including the transverse mesocolon, the patient was not included in this study. Gross evidence of celiac or peripancreatic node involvement, visceral metastasis noted at operation, or histological evidence of tumour extension up to the line of resection in the necropsy specimen were also causes for exclusion. Those with local involvement of the gastric serosa were considered acceptable for inclusion in the short-term survivor group. Those who died in the immediate postoperative period were excluded. All patients had evidence of death from gastric cancer on postmortem examination.

This study does not deal with operability, five-year survival or operative mortality rates, since it is purely a selective comparison. These matters embodying some of this material are being reported elsewhere.

The average age was not dissimilar in the two groups, being 62 years in the long-term and 63 years in the short-term survivors. The sex incidence in this disease is

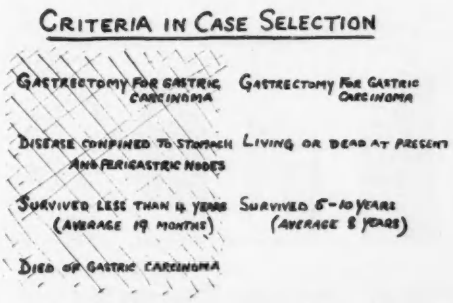


Fig. 1.—The criteria employed in selection of cases for comparison. Shaded area represents short-term survivors; clear area represents long-term survivors.

*Presented at the Annual Meeting of the British Columbia Surgical Society at Harrison Hot Springs, May 5-7, 1960.

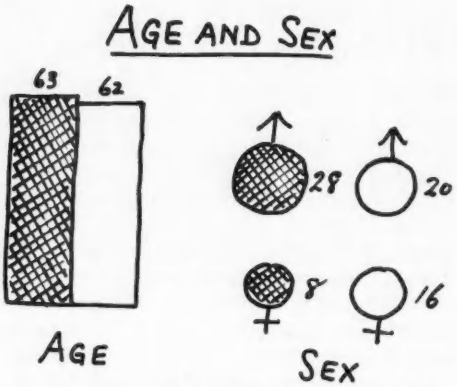


Fig. 2.—Comparison of age and sex incidence in whole numbers of short-term survivor group (shaded areas) and long-term survivor group (clear areas).

generally reported to be about 2.5 men to one woman. The comparative hardiness of women with respect to gastric cancer is reflected in both groups. Among the long-term survivors the ratio was 20 men to 16 women, and in the short-term group it was 28 men to eight women (Fig. 2).

Pain was the most frequent presenting symptom and was noted by 21 of the long-term and 26 of the short-term survivors. Next came weight loss with an incidence of 19 in each group. A close third was vomiting which affected 16 of the long-term and 18 of the short-term survivors. Less frequent symptoms with their respective incidences in the long-term and short-term survival groups included loss of appetite, 10 and 12; indigestion, 14 and 12; weakness, six and 10; blood in stools, three and four, and bloody vomitus, one and three.

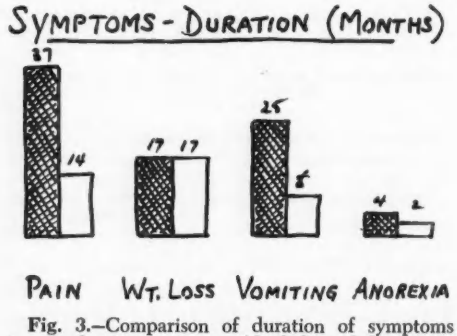


Fig. 3.—Comparison of duration of symptoms in months in short-term (shaded) and long-term (clear) survivor groups.

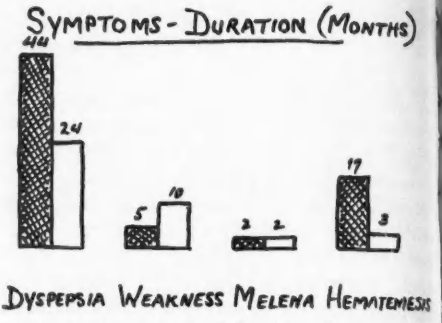


Fig. 4.—Comparison of duration of symptoms in months in short-term (shaded) and long-term (clear) survivor groups.

The duration of these symptoms was of some interest (Figs. 3 and 4). With the exception of weakness the reported duration of symptoms was shorter among the long-term survivors than among the short-term survivors. This finding supports the studies of Barclay,[1] but is directly opposed to those of Swynnerton[4] which uphold Macdonald[3] in his philosophical concept of biological predeterminism as a limiting factor of curability. The duration of symptoms in months, first with respect to long-term then to short-term survivors was as follows: pain, 14 and 37; weight loss, 17 and 17; vomiting, eight and 25; loss of appetite, two and four; indigestion, 24 and 44; weakness, 10 and five; bloody stools, two and two, and bloody vomitus, three and 17.

On physical examination abdominal tenderness was elicited in four of the long-term and 11 of the short-term survivors; an epigastric mass was discovered in four of the former group and in 10 of the latter. The laboratory findings were roughly comparable in the two series. Occult blood was found in stools of 27 of each group. Achlorhydria was present in 24 of the long-term and 27 of the short-term survivors. The sedimentation rate was raised in 26 of the long-term and in 30 of the short-term survivors. In the long-term survivor group only 13 had a hemoglobin reading of less than 80% and three had leukocytosis, whereas in the short-term survival group these figures were 11

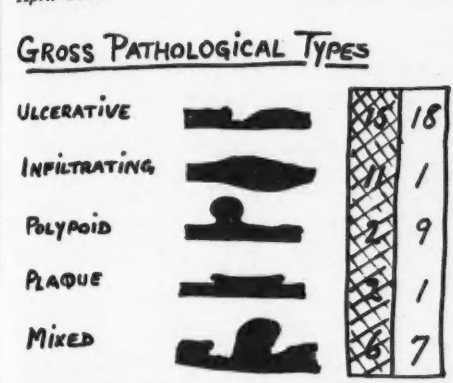


Fig. 5—Incidence in whole numbers of the various gross pathological types of tumour compared. Shaded area represents short-term survivor, and clear area long-term survivor group.

and two respectively. Radiographic studies with contrast media in the long-term survivors were positive in 21, doubtful in eight and negative in seven. In the short-term survivors these figures were 19, 13 and four respectively.

The site of the primary growth in the stomach was comparable in the two series. In the long-term survivors it was located in the cardia in three cases, the body in eight, pylorus in 23 and involved the entire stomach in two instances. Among the short-term survivors the site of the primary growth was the cardia in five, the body in 10, the pylorus in 20 and the entire stomach in one case.

The incidence of different gross pathological tumour types was notable as shown in Fig. 5. Those with polypoid cancers were predominantly long-term survivors in the ratio of 9 : 12, while those with infiltrating growths were predominantly short-term survivors in the ratio of 11 : 1. Ulcerative tumours were the most common in each group, affecting 18 of the long-term and 15 of the short-term survivors. Mixed types were next in frequency, seven occurring in the long-term and six in the short-term survival series. A plaque was infrequent, only one such instance being found in the long-term and two in the short-term survivor groups. Carcinoma originating from a peptic ulcer occurred only once among the long-term but three times among the short-term survivors.

Histological evidence of neoplastic extension to perigastric lymph nodes and neoplastic penetration of the serosal coat of the stomach were less common among the long-term survivors, 15 and 10 respectively, than among the short-term survivors, 24 and 19 respectively, as might be expected (Fig. 6). That the former figures were as high as they were, was unexpected. There was no significant difference in the histological grading of the tumours in either group. Grade 3 and 4 tumours comprised 24 of the long-term and 25 of the short-term survivors. Grade 1 and 2 tumours comprised 12 of the long-term and 11 of the short-term survivors. Excessive mucoid production was seen in only one of the long-term survivors but appeared in seven of the short-term survivor group.

The lymphocytic response of the stomach and perigastric lymph nodes was compared in the two series. The histological patterns described by Black, Opler and Speer[2] as being significant locally in the stomach, namely diffuse lymphocytic infiltration and follicular reaction; and in the perigastric lymph nodes, namely sinus histiocytosis and follicular reaction, were each assessed blindly by a competent pathologist, as to their extent. Grade 1 represents a mild degree and grade 4 an extreme degree of these various processes (Figs. 7 and 8). A summation of these counts for each process showed a significantly increased lymphocytic infiltration of the affected stomachs and follicular reaction of the perigastric lymph nodes among the long-term survivors, 84 and 60 respectively,

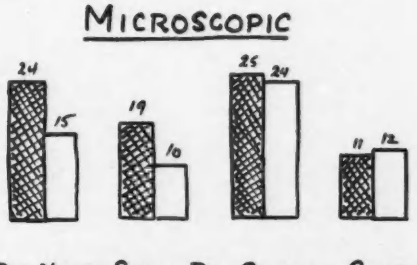


Fig. 6.—Comparison of the incidence in whole numbers of perigastric lymph node involvement and serosal penetration by tumour; also histological grading of the tumour. Shaded areas denote short-term and clear areas long-term survivors.

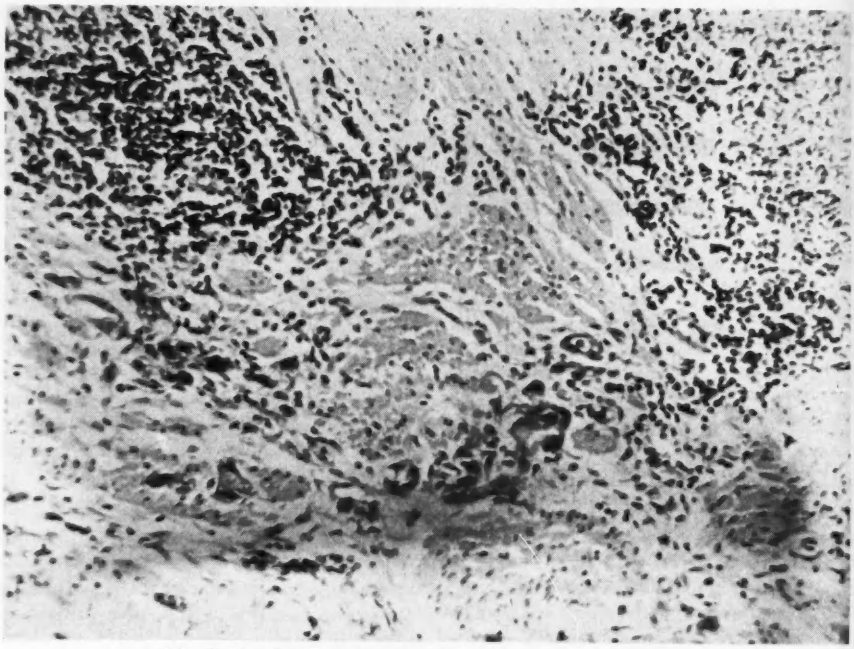


Fig. 7.—Grade 2 lymphocytic infiltration of primary tumour.

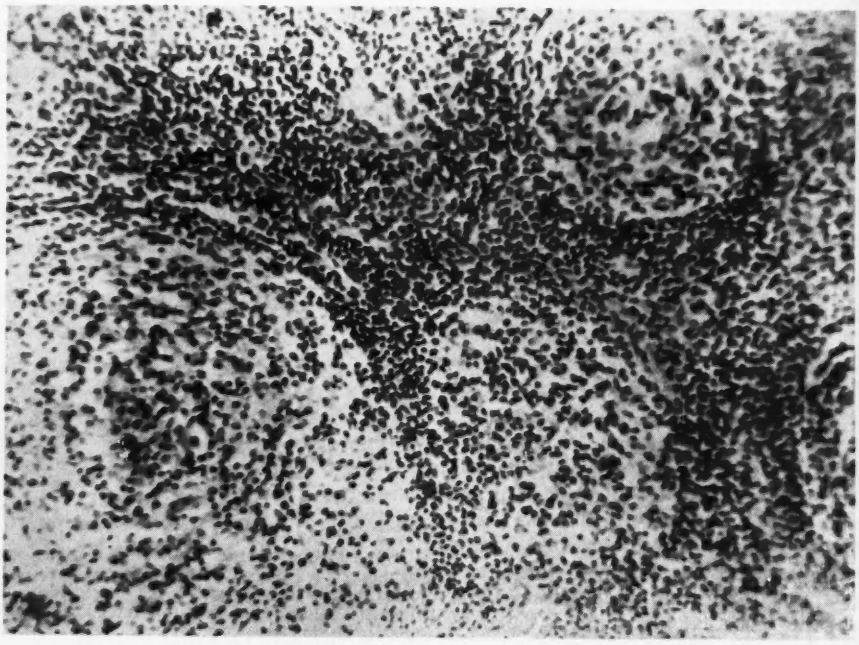


Fig. 8.—Grade 3 follicular reaction of perigastric lymph node.

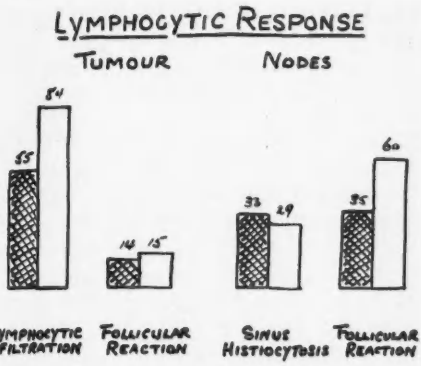


Fig. 9.—Comparison of lymphocytic response of primary tumour and perigastric lymph nodes in short-term survivor (shaded areas) and long-term survivor (clear areas) groups. Figures denote the sum of activity, graded from 0-4, of all cases with respect to each process.

compared to the short-term survivors, 55 and 35 respectively (Fig. 9). There was no real difference in the follicular reaction of the stomachs of the long-term and short-term survivors, 15 and 14 respectively, or of sinus histiocytosis in the perigastric lymph nodes, 29 and 33 respectively.

Summary

Clinical and pathological features of 36 persons who survived five or more years following gastric resection for carcinoma of the stomach are compared with similar features in an equal number of selected persons who succumbed in less than four years under similar circumstances.

Women had a greater natural resistance than men to the progress of this disease.

The duration of symptomatology was significantly longer in the short-term than in the long-term survivor group.

The presence of abdominal tenderness and the discovery of an abdominal mass was twice as common in the short-term as in the long-term survivors. Polypoid growths predisposed to long-term survival and infiltrating growth to short-term survival.

The production of excessive mucoid material by the cancer cell was a bad prognostic sign.

Gastric cancers arising in pre-existing benign peptic ulcers were associated with a poor prognosis.

Whereas tumour extension to the perigastric lymph nodes and gastric serosal penetration by the primary growth were more common in the short-term survival group, these features should not preclude attempts at complete surgical ablation since they were present in 40% and 27%, respectively, of the long-term survivor group.

There was a significantly increased lymphocytic response in the long-term survivor group as evidenced by increased diffuse lymphocytic infiltration of the stomach wall and increased follicular hyperplasia in the regional lymph nodes.

Acknowledgments

I am grateful to Dr. L. T. Maxwell for his assistance in gathering material and to Dr. H. K. Fidler for the histological interpretation of lymphocytic response and tumour grading.

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Résumé

Dans cet article, l'auteur étudie 36 cas de malades survivant au moins cinq ans à une résection gastrique pour cancer, comparativement à un groupe de même importance n'ayant pas présenté une survie de plus de cinq ans. Il ressort de là que les femmes offrent une plus grande résistance au progrès de la maladie que les hommes. L'apparition des symptômes remontait à beaucoup plus longtemps chez ceux qui survécurent le moins. La découverte de masses intra-abdominales ne signifie pas grand chose. Sont de mauvais pronostic les faits suivants: une croissance tumorale par infiltration; l'hyperproduction de substances mucoïdes; les carcinomes apparaissant sur un ulcère peptique primitivement bénin. L'envahissement des ganglions lymphatiques et l'infiltration de la séreuse gastrique sont un mauvais signe, mais ceci peut être modifié par l'importance de la résection. L'infiltration lymphocytaire diffuse de la paroi gastrique et l'hyperplasie folliculaire des ganglions régionaux correspondent aux cas à longue survie.

A NEW TECHNIQUE IN THE DIAGNOSIS OF HIRSCHSPRUNG'S DISEASE*

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HIRSCHSPRUNG'S disease may be defined on a histopathological basis as a congenital absence of the myenteric nerve plexuses extending from the anus for a variable distance proximally; the majority do not extend proximal to the sigmoid colon.

Although this disease was not unknown before his time, it was Hirschsprung[8] who described in 1888, the postmortem findings in two cases, noting that "when the abdomen was opened, two enormously dilated loops of large intestine presented themselves – the sigmoid and the even more severely distended transverse colon. Also, the other part of the large intestine appeared somewhat dilated; only the rectum was not enlarged, but rather seemed to be the site of some kind of slight narrowing".

In January 1898, Treves[16] reviewed the literature on idiopathic dilatation of the colon and described a typical case of Hirschsprung's disease in which he performed a colostomy and later removed the descending colon, sigmoid colon and most of the rectum.

Unfortunately, for many years, the pathogenesis of this disorder was sought in the distended bowel segment.

The history of the final evaluation of the pathology of Hirschsprung's disease is interesting. Intensive medical measures, spinal anesthesia and sympathectomy and partial or total colectomy were among the various forms of treatment employed. In August 1948, Swenson and Bill[12] reported a new method of treatment by resection of the lower rectal segment and as much of the affected bowel as might be necessary, with preservation of the sphincters. This procedure constituted a major advance in the treatment of this disease. It is often erroneously stated that Swenson was the first to concentrate upon the narrow and apparently normal distal segment, but Tittel[15] in 1901, Cameron[3] in 1928, and Robertson and Kernohan[11] in 1938, all claimed to have demonstrated scantiness, degeneration or absence of ganglion cells in the bowel wall.

Dalla Valle[4] in 1920 reported the finding of megacolon in two siblings, with absence of ganglion cells in the sigmoid colon and normal ganglia in the proximal bowel.

Tiffin *et al.*[14] in 1940, and Zuelzer and Wilson[17] in 1948, reported aplasia of ganglion cells of the myenteric plexus in the distal bowel segment.

Finally, Bodian, Stephens and Ward[1] clarified the various types of megacolon in such a way as to make intelligent selective treatment possible.

It is convenient, when discussing the clinical features of Hirschsprung's disease, to recognize two types. The first is that form which presents in the neonatal period, and the second, that which is encountered in the older infant or child.

To consider the latter group first, it must clearly be understood that the anomaly is congenital, and that on careful questioning it is always possible to elicit a history of bowel trouble, however insignificant, since birth. Usually the initial neonatal symptoms may be so minor or so transitory that hospitalization is not necessary: even medical advice from the practitioner may not be sought. Following the early episode of obstipation and/or abdominal distension and vomiting, there may be a relatively asymptomatic period lasting weeks or months until the infant is in need of occasional aperients, suppositories or washouts. Interspersed with this benign course, at any time, attacks of vomiting, abdominal distension, fever and diarrhea may occur. These constitute a serious complication and are usually loosely designated as "enterocolitis".

The severity of the symptoms is as a rule, with certain noteworthy exceptions, directly related to the length of the aganglionic segment.

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*Presented at Congress of South African Association of Surgeons, Durban, September 1960.

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Fig. 1.—Radiograph showing typical barium enema findings in a patient with long-segment Hirschsprung's disease.

Examination usually reveals an undersized child with a distended abdomen and an empty rectum.

The other group of patients present with symptoms of Hirschsprung's disease in the neonatal period, usually within a week of birth, with bile-stained vomiting, abdominal distension and constipation — all the features of a low small intestinal or large intestinal obstruction. It is often impossible in such cases to determine the nature of the obstruction on clinical grounds, and in these circumstances radiological investigation is obligatory. Unfortunately, in the neonatal infant it is impossible to distinguish dilated ileum from dilated colon, so that in every case in which the diagnosis of Hirschsprung's disease is entertained, an opaque enema must be performed before a bowel washout or any other preparation of the bowel is attempted.

In a typical case, a barium enema will clearly demonstrate the distal narrow, aganglionic segment (Fig. 1); the cone or funnel proximal to this segment, and the dilated, hypertrophied bowel yet further proximally. The diagnosis in such a case presents little difficulty. A delay of 24 hours or more in eliminating the contrast material also favours the diagnosis.[5]

Diagnostic difficulties are encountered, however, in those with short-segment Hirschsprung's disease in which the dilatation extends distally to involve the rectum. In such cases, additional diagnostic procedures are necessary to distinguish true Hirschsprung's disease from acquired megacolon or colonic inertia (Fig. 2).

In infants, too, radiological study is less satisfactory than in the older child as the characteristic changes take time to develop.

The diagnosis can be established by rectal biopsy. Adequate microscopic sections of the rectal wall in Hirschsprung's disease reveal the characteristic absence of ganglion cells in the myenteric plexus.[2]

The biopsy technique described by Swenson[13] involves removal of a 5 mm. x 10 mm. full-thickness section of the rectal wall and repair of the wall and mucosa



Fig. 2.—Short-segment Hirschsprung's disease.

with two layers of interrupted sutures. This procedure is carried out under general anesthesia, operating through a bivalve speculum, the lower end of the incision being 3 cm. from the mucocutaneous junction. The specimen so obtained is examined microscopically and a search is made for ganglion cells in the intermuscular nerve plexuses. The presence of ganglion cells in such a biopsy rules out a diagnosis of Hirschsprung's disease.

According to Bodian,[2] the diagnosis of Hirschsprung's disease can be made by examination of a biopsy of mucosa and submucosa only, by searching for ganglion cells in the submucous nerve plexuses. This submucous biopsy is carried out under general anesthesia and the mucosa is sutured, leaving a drain *in situ*. It does not involve excision of any portion of the muscular wall.

A third biopsy technique, described by Hiatt,[7] involves an approach to the posterior rectal wall through an incision in the natal cleft midway between the anus and the coccyx. The biopsy specimen is obtained without entering the bowel lumen, thus avoiding contamination by rectal contents. General anesthesia is required for this procedure.

The fact that such a selection of biopsy techniques exists, in itself suggests that a completely satisfactory procedure has not yet been devised. Experience in the pathology department of the Red Cross Children's Hospital in Cape Town, indicates that it is difficult or at times impossible to base any sound conclusions on the micro-

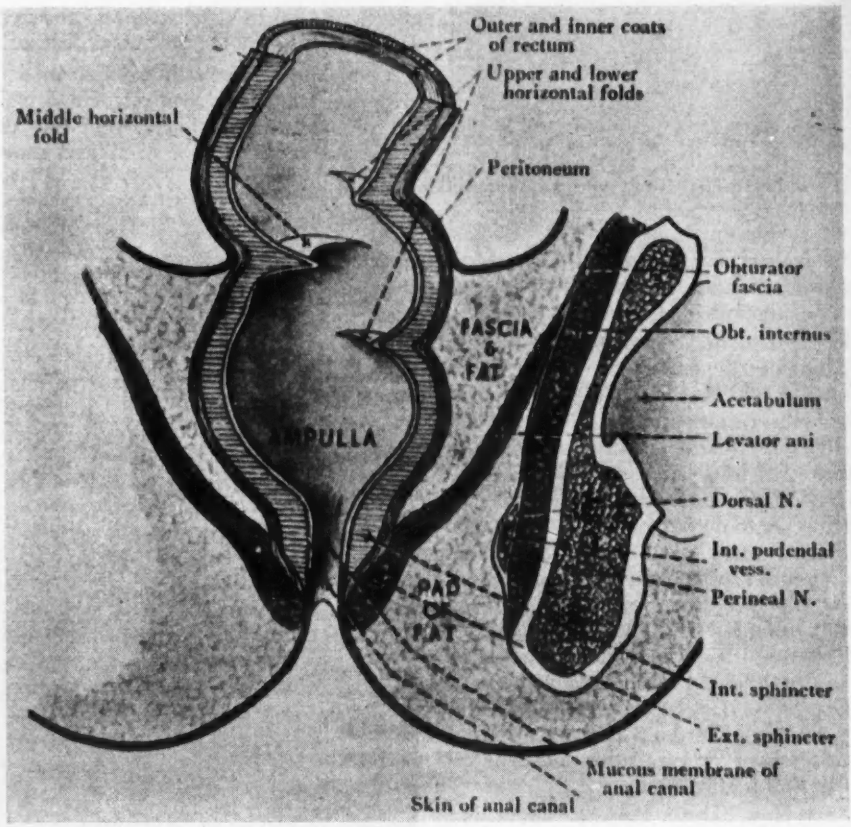


Fig. 3.—Diagrammatic coronal section through pelvis indicating the valves of Houston which consist of mucosa and muscle. (Reproduced from Jamieson's Atlas of Anatomy by kind permission of the publishers, E. & S. Livingstone Ltd., Edinburgh.

scopic findings in submucous biopsy specimens obtained by the technique described by Bodian.

The biopsy procedures described by Swenson and Hiatt are by no means innocent operations without ill effects, and in our experience have, at times, been followed by serious rectal bleeding, sepsis and perirectal or retrorectal fibrosis. The latter greatly interferes with subsequent operations in the region of the anal canal and rectum. These undesirable features of the existing methods prompted a search for a more satisfactory biopsy technique.

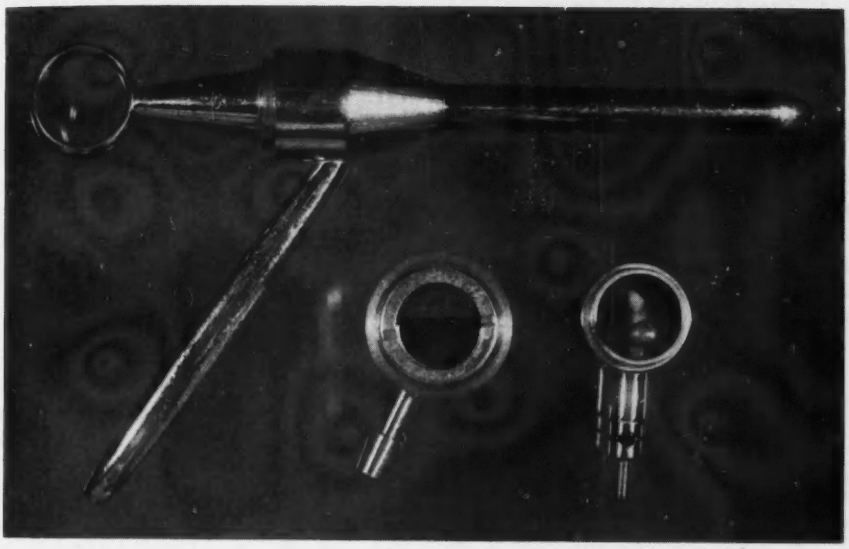


Fig. 4a

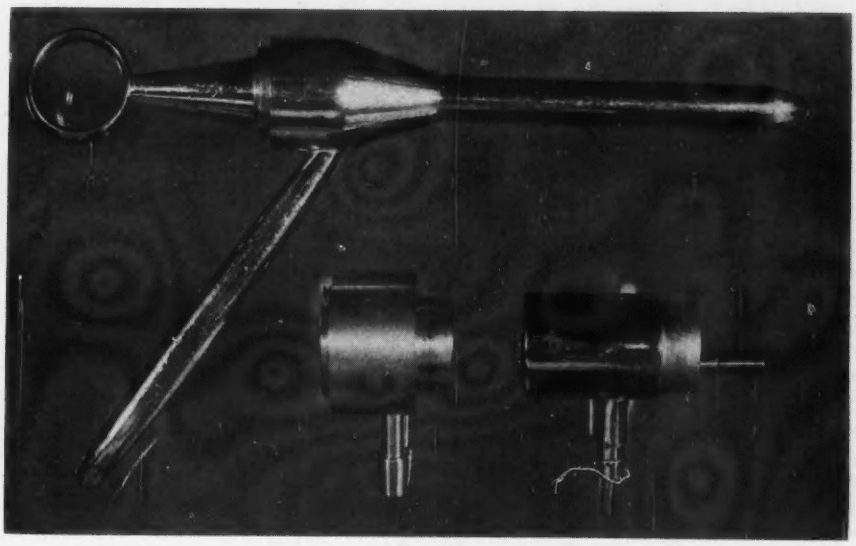


Fig. 4b

Figs. 4a and b.—Infant sigmoidoscope with light carrier and eyepiece with renewable rubber washer.

The three transverse folds or valves in the rectum, originally described by Houston,[9] are illustrated in Fig. 3, with the lowest and highest on the left side, and the middle one on the right. Occasionally a fourth valve is present. In adults, the lowest valve of Houston is about 5 cm. from the mucocutaneous junction; in children this distance varies from approximately 1 cm. to 3 cm. depending on the age of the child. According to Gabriel[6] the curves of the rectum are accentuated on their concave aspects by infoldings of the whole thickness of the rectal wall which constitute the rectal valves of Houston. Hughes[10] also maintains that the valves of Houston contain extensions from both muscle coats and fibrous connective tissue, an observation that is confirmed in many textbooks of anatomy and rectal surgery. Houston[9] himself described the presence of circular muscle fibres in these valves.

Consideration therefore was given to the feasibility of obtaining a suitable biopsy specimen from one of the valves, which would include sections of mucosa and muscle tissue. Such a procedure should involve minimal trauma, little or no risk of perforation of the rectal wall and should be performed on patients of all ages, without the use of general anesthesia. The biopsy specimens so obtained should be adequate to demonstrate the absence of ganglion cells, of diagnostic significance in patients with Hirschsprung's disease.

After considerable investigation on a trial-and-error basis, it was concluded that optimum sedation for this procedure was provided by a combination of Seconal sodium,® gr. ¾ per stone (14 lb.) of body weight and chlorpromazine hydrochloride, 2mg./lb., administered orally, one and one-half to two hours before the operation.

Additional clinical trials led to the conclusion that the most effective means of rectal cleaning in preparation for this biopsy procedure is accomplished by the insertion of one Dulcolax® suppository in the rectum, one hour preoperatively, the patient emptying his bowel before the operation.

After such preparation and under sedation, the patient, who is usually in a deep sleep, is placed on the operating table in a prone, jack-knife position. Final rectal cleaning is facilitated by suction with a bronchoscope sucker. Any of the child sigmoidoscopes can be used (Figs. 4a and b); but at the Red Cross Children's Hospital, Cape Town, the Lloyd-Davies infant sigmoidoscope with proximal lighting is considered preferable for this procedure in children of any age. After insufflation of the rectum the folds of Houston stand out prominently but on removal of the eyepiece of the sigmoidoscope to permit insertion of the biopsy forceps, the rectum collapses and the folds are not readily recognizable. To overcome this difficulty a special eyepiece was designed for the sigmoidoscope, incorporating a renewable rubber washer which prevents the escape of air when the biopsy forceps is inserted, and permits the performance of biopsy while rectal insufflation is maintained. If such an eyepiece is not available the shelf-like fold of the rectal valve to be biopsied can be maintained as a prominent projection into the lumen by packing a swab beneath it.

It was found to be advisable to remove three specimens from the valve to provide adequate biopsy sections. Some bleeding may occur but this is usually minimal and requires no special care.

The size of the biopsy specimen is important. If the "bite" is too small it will remove mucosa only, especially if the valve is not standing out prominently when the section is taken. It should be possible to demonstrate the definite presence or absence of ganglia in every case, provided the amount of biopsy material removed is adequate (Figs. 5a and b). Three sections should be cut from each tissue fragment, thus tripling the total biopsy area available for microscopic examination.

This method of rectal biopsy was performed on 40 patients of whom eight were children or newborn infants with Hirschsprung's disease. The diagnosis was confirmed by histopathological study of surgical excised portions of bowel in all but one of the latter group; the remaining patient presented typical clinical and radiological features of Hirschsprung's disease but was not subjected to operation. Biopsies were performed by this technique on 32 children

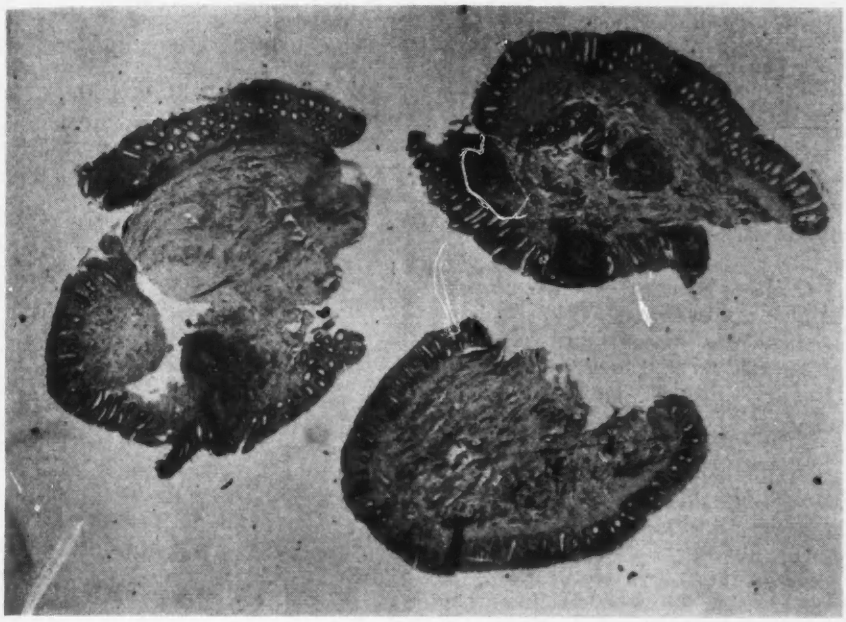


Fig. 5a

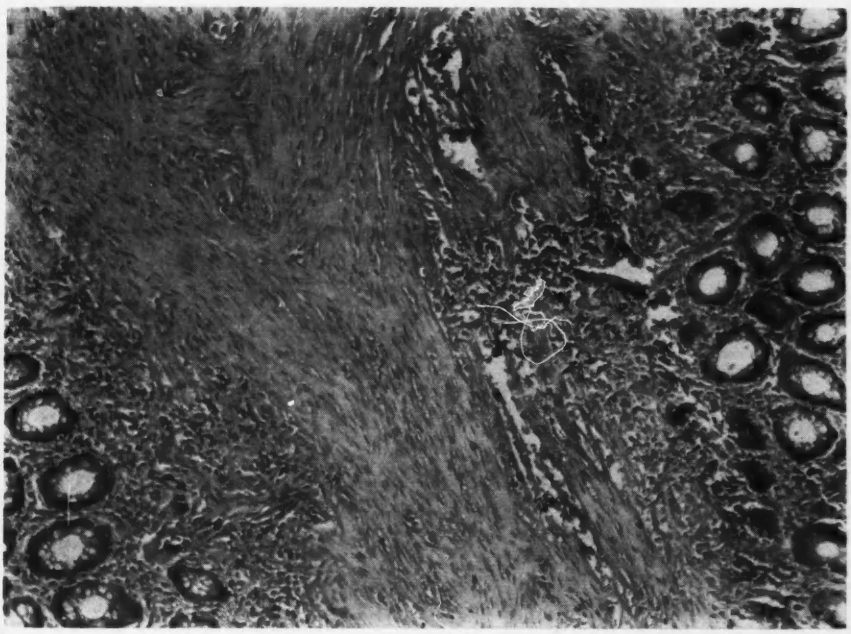


Fig. 5b

Figs. 5a and b.—(a) Material obtained by biopsy of Houston's valve (original magnification x 35). (b) High-power view showing membrane with muscle between (original magnification x 150).

with burns, osteitis, herniae and other unrelated disorders, as a control group. In the latter group, ganglion cells were seen in every case in which an adequate biopsy specimen was taken. No ganglion cells were observed in one specimen that consisted almost entirely of mucosa, but repeat biopsy on the same patient revealed the presence of normal ganglion cells.

The obvious importance of rectal biopsy in the diagnosis of Hirschsprung's disease was emphasized by one case in particular. This patient was a boy who presented typical clinical and radiological features of this disorder (Fig. 6), and who had a sister with a proven diagnosis of Hirschsprung's disease. Under these circumstances rectal biopsy was considered unnecessary to establish the diagnosis but this procedure was carried out nevertheless, and to the surprise of all concerned, the specimen revealed the presence of ganglion cells in the rectal wall. Subsequent examination of the colon and rectum confirmed the fact that this patient did not have Hirschsprung's disease.



Fig. 6.—Barium enema radiograph of patient with a clinical syndrome resembling Hirschsprung's disease.

Summary

The pathology and clinical types of Hirschsprung's disease (congenital megacolon) are discussed and the diagnostic criteria are considered from the clinical, radiological and histological points of view.

The most conclusive criterion for diagnosis to date is the demonstration of complete absence of ganglion cells in the submucosal and intermuscular nerve plexuses of the distal colon and rectum.

Disadvantages of previously described techniques of rectal biopsy are discussed and a new simplified method for obtaining biopsy material from Houston's valves is described.

This technique does not require general anesthesia, does not involve incision of the rectum or suture of the mucosa, does not result in sequelae that interfere with a subsequent Swenson or Duhamel operation, and has not been complicated by hemorrhage, sepsis or fibrosis.

In a series of 40 biopsies using this technique, ganglion cells were demonstrable in every child who did not suffer from Hirschsprung's disease, and were absent in every patient with an established diagnosis of this disease.

Adequate biopsy of a valve of Houston can provide unequivocal evidence of the presence or absence of Hirschsprung's disease.

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Résumé

La maladie de Hirschsprung peut être définie comme étant une absence congénitale des plexus nerveux myentériques, depuis l'anus en remontant vers le haut du tube digestif; toutefois, dans la majorité des cas, cette absence ne dépasse pas le côlon sigmoïde. C'est ainsi que se forme le syndrome du megacôlon.

Après avoir passé en revue la littérature sur ce sujet, l'auteur aborde le problème du diagnostic. Le critère le plus certain pour confirmer une maladie de Hirschsprung est évidemment la constatation sur coupes histologiques de l'absence complète de cellules nerveuses ganglionnaires dans la sous-muqueuse et entre les faisceaux musculaires lisses de l'intestin. Il a donc été préconisé de pratiquer des biopsies de la paroi rectale. Cette technique a cependant des désavantages et des inconvénients qui sont discutés en détail.

L'auteur propose une nouvelle méthode. Il préconise à cette fin d'utiliser les valvules rectales en lieu et place de la muqueuse rectale proprement dite; ceci a l'avantage de permettre l'obtention biopsique facile de fragments contenant de la sous-muqueuse et de la musculaire. Toute anesthésie générale devient inutile, de même que toute incision et toute suture. Cette technique a déjà été employée chez 40 malades avec des résultats satisfaisants.

En conclusion, il semble que la biopsie d'une des valvules de Houston permette un diagnostic précis et aisé de la maladie de Hirschsprung.

QUALITIES NEEDED BY THE SURGEON*

Those special abilities which are needed above all others by the successful surgeon, have been detailed excellently by the English physician Hutchinson, in a talk to students of the London Hospital Medical College. He called them the "seven presents which heaven should give to a future surgeon."

First of all, the surgeon must have a sound and healthy constitution, one which is able to resist fatigue and contagion.

Second, the surgeon needs good fortune to attend his efforts. This gift is rare enough, and when it is lacking, hard work and patience may compensate for what is absent.

Third would be intelligence, but not too much of it. Intelligence needs to be tempered to a certain degree by great zeal.

Fourth is a considerable endowment of equanimity, which will enable the surgeon to master difficult situations and withstand the onerous burdens of daily troubles and problems.

Fifth, Hutchinson proposes a sense of justice toward patients as well as toward the surgeon's staff and toward himself.

Sixth, he mentions an appreciation of beauty; since any disease is a contradiction of beauty, the surgeon ought to be able to observe, recognize and enjoy beauty outside his everyday hospital work.

Finally, the surgeon should be a man of much good humor. Good humor will support him in withstanding extravagant demands not only on the part of his patients but also on the part of their relatives. A few words of good humor often will help more than psychotherapeutic efforts.

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THE SURGERY OF THE THORACIC INLET*

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THE THORAX is bounded by the thoracic inlet above, and the diaphragm below. Over the past 25 years the lower boundary, the diaphragm, has presented no serious surgical barrier between the abdomen and the thorax. As Sir Heneage Ogilvie, that coiner of apt phrases, has so well put it, "the iron curtain of the diaphragm" no longer exists. At the other end of the thorax, the thoracic inlet has proved a somewhat more difficult problem. This is because of its small size, bounded as it is by the upper thoracic vertebrae, the first rib, the clavicle and the manubrium sterni. Access is therefore limited. Through it go certain important structures which may be of concern to the surgeon, such as the trachea and esophagus, the subclavian vessels, the carotid vessels, the sympathetic chain, the first thoracic nerve, and the thoracic duct. Furthermore, the roots, trunks and the divisions of the brachial plexus enter into this field. Pathologic entities which may call for thoracic inlet exploration may be found in connection with the thyroid gland, for example a retrosternal goitre, and with the parathyroid glands, in the form of a parathyroid adenoma. Cervical rib, osseous or fibrous, may involve the brachial plexus. Aneurysms or obliterative disease of the subclavian, carotid and vertebral arteries may necessitate exploration of this area. Cystic hygroma may spread through the thoracic inlet. Tumours may develop on the first thoracic nerve. Enlargement of the upper mediastinal lymph nodes may occur due to a variety of causes and these can be reached from the neck via the thoracic inlet.

The anatomy and surgical approaches to this area are probably less familiar to surgeons than is the case with the lower thoracic outlet. Therefore, it appears useful to review the surgical approach to this area. To reach midline or near midline structures situated anteriorly, the sternum splitting incision is useful and in these days of openheart surgery this approach is being widely used. For more laterally placed structures, however, and particularly in the approach to the lower half of the brachial plexus and the sympathetic chain, the supraclavicular approach of Telford is to be preferred. It is perfectly feasible to divide the clavicle and thus open up the area somewhat, but this should rarely be indicated inasmuch as this bone lies in a plane anterior to the first rib, and it is the restricting curve of the first rib which so often provides the difficulty of access.

Of recent years the author has had occasion to explore the thoracic inlet for a wide variety of conditions, some of which are set out as follows.

1. Lesions requiring cervico-dorsal sympathectomy:
   (a) Raynaud's disease
   (b) Hyperhidrosis
   (c) Angina pectoris
   (d) Disciform macular degeneration of the retina.
2. Neurofibroma of the first thoracic nerve.
3. Cervical rib, both osseous and fibrous, causing pressure on the lower brachial plexus.
4. Aberrant phrenic nerve causing compression of the subclavian vein.
5. Cystic hygroma.
6. Aneurysms of the first and second intercostal arteries.
7. Axillary artery aneurysm requiring proximal control.

The key to understanding of the anatomy and exploration of this area is provided by the anterior approach to the cervico-dorsal sympathetic chain. This was described in detail by the author in 1957,[1] and the reader is referred to this article for a detailed description of the technique involved. Perhaps in no area of the body is minute attention to detail more important, in order to provide a clear and adequate

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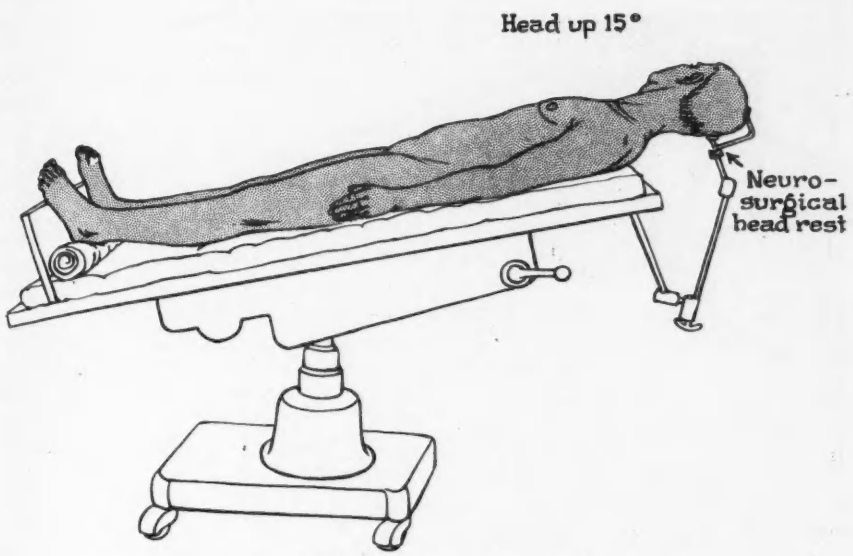


Fig. 1.—Position of patient for exploration of thoracic inlet.

exposure. The technique of the exposure is illustrated in Figs. 1, 2, 3 and 4.

The Following Special Points Should be Observed

1. The patient should be carefully positioned on the operating table (Fig. 1). The table is tilted, head up, about 10° because this decreases venous congestion. The head is turned to the opposite side, and the shoulder and arm are pulled well down and fixed thus to depress the clavicle out of the field.
2. The incision is short, not more than three inches in length, and is sited over the clavicular head of the sternomastoid muscle. It is unwise to incise lateral to this muscle, because a mass of fat and a friable plexus of supraclavicular veins are thereby exposed in the supraclavicular fossa (Fig. 2).
3. The clavicular head of the sternomastoid muscle is divided close to the clavicle by first passing a right-angled hemostat beneath it, and elevating the muscle, before it is divided with a scalpel (Fig. 3).
4. The internal jugular vein is retracted medially and at this stage the position of the scalenus anticus muscle is felt as a taut, broad band. The areolar tissue overlying the muscle is stroked aside by a small gauze sponge and thus the phrenic nerve is located. The nerve is dissected free from the fascia of the muscle and is retracted medially (Fig. 4).
5. The scalenus anticus muscle is isolated by carefully passing a right-angled hemostat beneath it. It is then divided by a scalpel. This should be done cautiously because a very short branch of the subclavian artery may enter the deep surface of the muscle. This vessel is easily torn away from the subclavian artery causing undesirable arterial hemorrhage. The scalenus anticus muscle should always be divided in any exploration of the area because this maneuver greatly improves the exposure.
6. The subclavian artery is thus clearly exposed in its three parts. This vessel must be freed completely. This is facilitated by dividing the thyroid axis artery; care must be taken, however, not to divide the vertebral artery (Fig. 5).
7. Sibson's fascia is then divided by gently nibbling through it piecemeal, exposing the extrapleural fat. The pleura is then

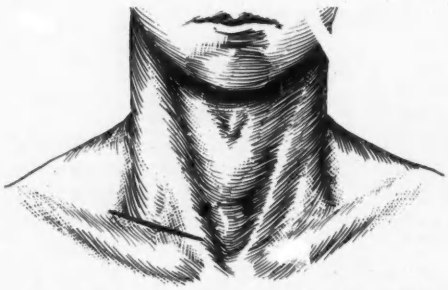


Fig. 2.—Site of incision for exploration of thoracic inlet or upper dorsal sympathectomy by the anterior route.

stroked down off the ribs and thoracic vertebral bodies by the index finger. This maneuver should be carried out in any full exploration of the area to improve the exposure and display the whole of the inner rim of the first rib (Fig. 5).

8. The sympathetic chain is then felt overlying the neck of the first rib. It is easier to locate the chain at first by touch rather than by sight. Further laterally the first thoracic nerve is both felt and seen (Fig. 6). Two other approaches to the cervico-dorsal sympathetic chair are available:
   (a) The posterior approach of Adson
   (b) The axillary approach of Atkins.[2]

However, the most universally applicable approach is the anterior one described above because it permits ample exposure of the upper dorsal sympathetic chain as far down as the fourth thoracic ganglion, as well as the stellate ganglion. In addition it permits a very thorough exploration of the thoracic inlet so that any unsuspected lesion such as a fibrous cervical rib will not be overlooked. This particular approach was used in operations on all of the conditions listed earlier. It would therefore be wise for surgeons to familiarize themselves with this anterior approach because of its wide applicability for a variety of conditions in addition to those requiring operations on the sympathetic chain.

It is not proposed to discuss the surgery of the sympathetic chain for Raynaud's disease as this has been described by many authors in a number of publications. Furthermore, the author has encountered

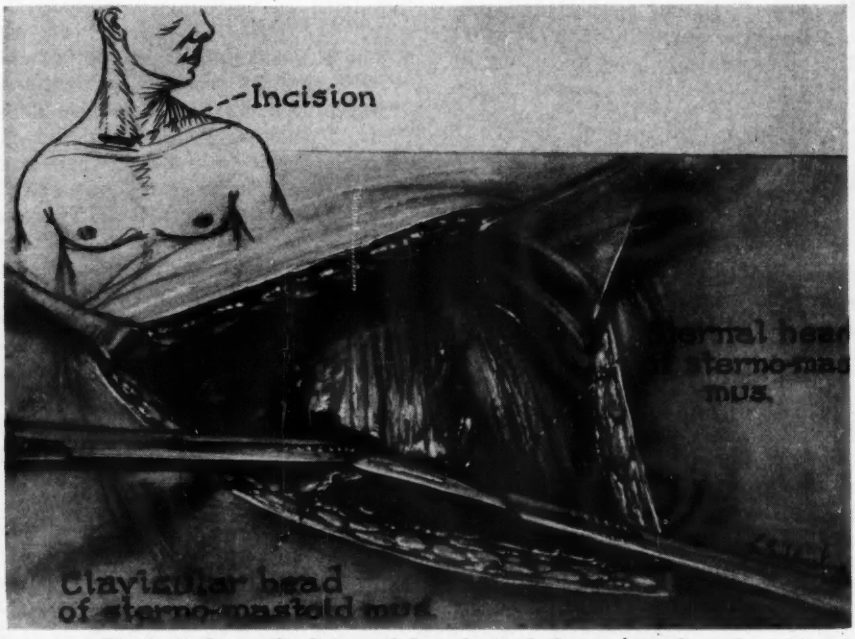


Fig. 3.—Technique for division of clavicular head of sternomastoid muscle.

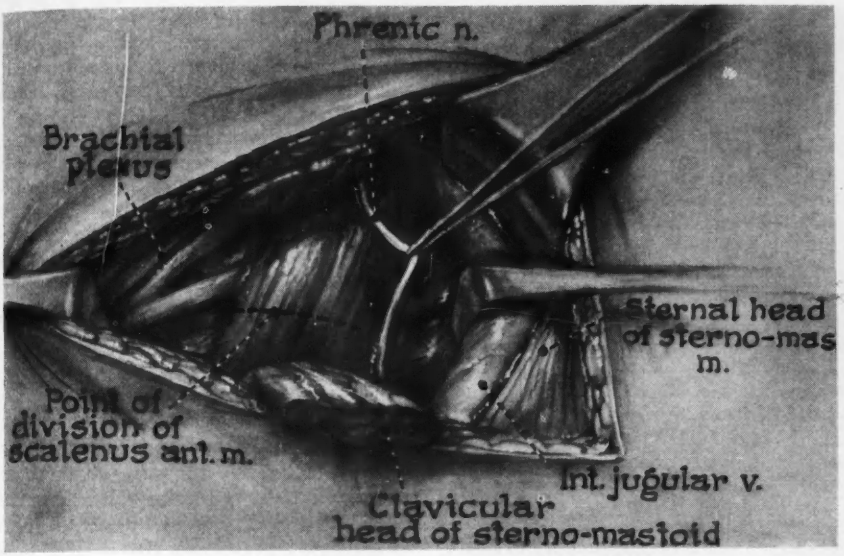


Fig. 4.—Display of scalenus anticus muscle before division.

relatively few patients with Raynaud's disease in Canada as compared with its frequency in Great Britain. It is interesting to speculate as to why this is so, since Canada has severe winters. One possible explanation lies in the universal use of central heating in Canada. Thus, Canadians are warm 90% of the time whereas the inhabitants of Great Britain are cold 90% of the time!

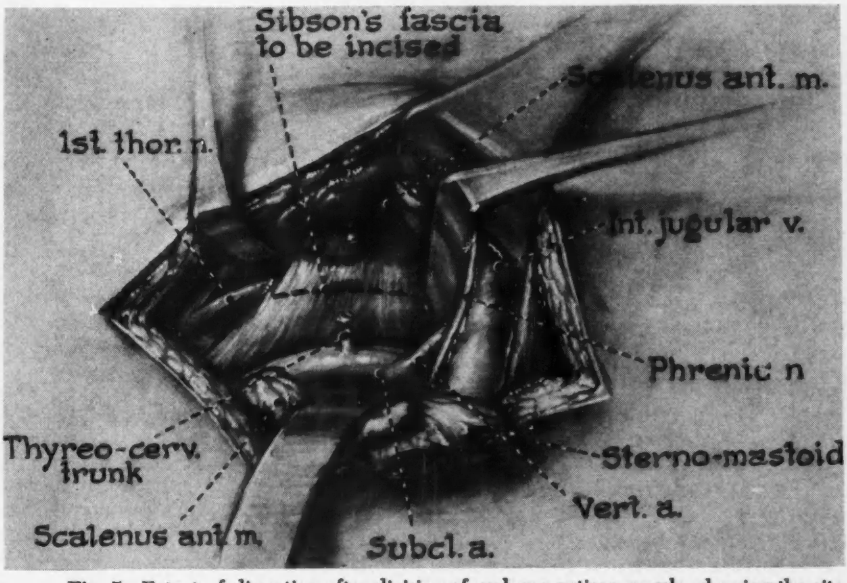


Fig. 5.—Extent of dissection after division of scalenus anticus muscle, showing the site of division of Sibson's fascia.

Hyperhidrosis

CASE 1.—Mrs. W., aged 31 years, had suffered from excessive sweating of her hands all her life. This was made worse by excitement. Her hands would frequently drip with perspiration and of late she had found it hard to drive her automobile because the steering wheel became so slippery due to this excessive sweating. She also suffered from eczema of her hands. Radiography revealed bilateral cervical ribs (Fig. 7) and it was considered that these possibly might be an initiating factor in producing the hyperhidrosis. It was also thought that the eczema might be improved by rendering her hands dry. Therefore bilateral excision of the cervical ribs was carried out together with removal of sympathetic ganglia T2 and T3. The stellate ganglion was carefully preserved. Following this operation her hands remained dry and she experienced no more sweating; the eczema however was not improved and indeed seemed somewhat worse. Now she has difficulty driving her automobile because her hands are so dry that they tend to slip on the steering wheel.

CASE 2.—Mrs. I.C., aged 39 years, had a history of excessive sweating of her hands and feet since birth. In 1944 she had a bilateral lumbar sympathectomy and a right upper dorsal sympathectomy which was carried out by the posterior approach. After this operation, however, the sweating of the left hand became more pronounced to the extent that perspiration would drip from the finger tips. In 1959 the author performed an upper dorsal sympathectomy from the anterior approach. The second, third and fourth thoracic ganglia and the lower stellate ganglion were excised. This produced a dry hand but also a Horner's syndrome.

Angina Pectoris

Upper dorsal sympathectomy was formerly used for the control of pain in angina pectoris. This procedure is now rarely indicated in view of the efficacy of revascularization procedures for the myocardium. However, in the following case the circumstances were such that it did seem to be indicated.

CASE 3.—Mr. A.C., aged 62 years (patient of Dr. J. C. Dundee), had had Charcot-Marie-Tooth atrophy for 17 years and an

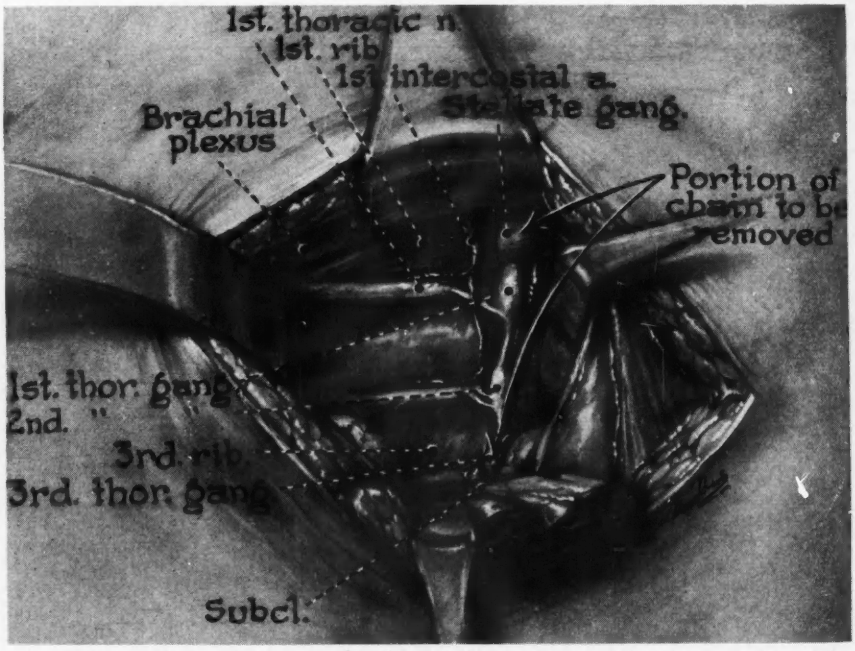


Fig. 6.—Display of the upper dorsal sympathetic chain and its relations, achieved by the anterior approach.

ulnar palsy of the right hand for 30 years due to an accident. He had had angina pectoris of increasing severity for the past 17 years. This had been much worse over the past three years during which he had six admissions to hospital and at the time of operation was in status anginosis. Therefore, a left upper dorsal sympathectomy was done with removal of ganglia T1—T4, and the stellate ganglion. In the postoperative period he developed posterior inferior cerebellar artery insufficiency and a further myocardial infarction, from all of which he recovered. He was relieved of his anginal pain in the precordium and down the left arm. He still had angina in his right arm, but it was bearable without narcotics. He died one year later due to myocardial infarction.

Had this man been in better condition, a further right upper dorsal sympathectomy would likely have relieved him of his angina on the right side. No direct cardiac surgery could be contemplated in this case, and therefore the pain-relieving operation of upper dorsal sympathectomy seemed to be indicated.

Disciform Macular Degeneration

Case 4.—Mr. C. (patient of Dr. S. Drance) had lost the central vision of his right eye from disciform macular degeneration due to vascular insufficiency. The left eye was beginning to develop a similar condition. Therefore, in the hope of increasing the blood supply to the left retina, a left stellate ganglionectomy was performed. Unfortunately, the desired increase of vascularity of the retina did not occur.

Neurofibroma of the Right First Thoracic Nerve

Case 5.—Mrs. L.B. (patient of Dr. L. McConnell), aged 19 years, had a routine chest radiograph which showed a round shadow in the right thoracic inlet area (Figs. 8 and 9). Her only symptoms were occasional weakness and numbness of the right forearm. Detailed radiographs showed erosion of the right pedicle of the 1st thoracic vertebra. A myelogram was normal. The right thoracic inlet was explored. A golf ball size tumour was found arising from the eighth cervical nerve. Frozen section showed it to be a neurofibroma. Therefore the capsule was incised and it was removed by morsellation. The eighth cervical nerve was sacrificed because it passed directly through the tumour. No neurological deficit resulted and the former slight symptoms were relieved.

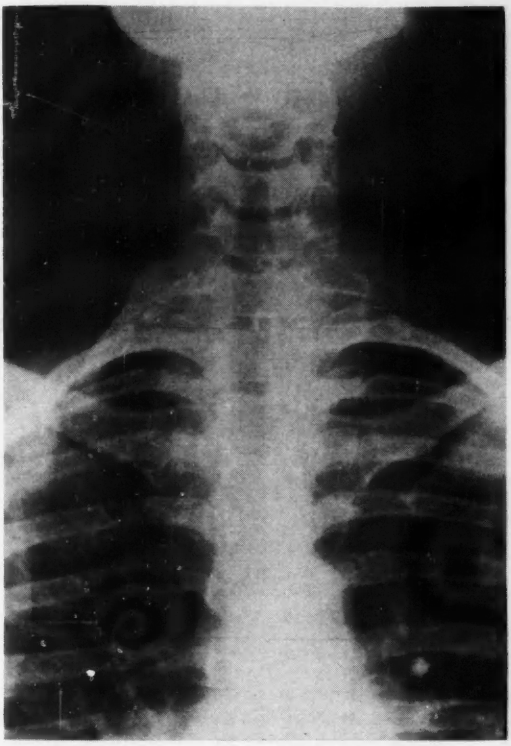


Fig. 7.—Mrs. W. Chest radiograph showing long cervical rib on right and rudimentary one of the left.

Cystic Hygroma

Case 6.—M.N., a girl aged two years and eight months, had a fluctuant lump the size of a plum in the left side of the neck, for one year. It was excised by the family doctor and was reported to be a semisolid cystic hygroma. It recurred in four weeks and further attempt at excision was made but because of severe bleeding the operation was abandoned. When seen by the author the child had an obvious fluctuant swelling in the left supraclavicular area, which increased in size on crying. A chest radiograph showed an extensive shadow in the left upper zone (Fig. 10). This, therefore, was an extensive cystic hygroma which extended down through the thoracic inlet into the upper mediastinum and left upper thorax. For this reason excision was performed in two stages. The first operation consisted of an exploration of the left thoracic inlet. This disclosed an extensive racemose lymphangioma infiltrating up the neck, across to the right side behind the esophagus and down through the thoracic inlet to the arch of the aorta. The

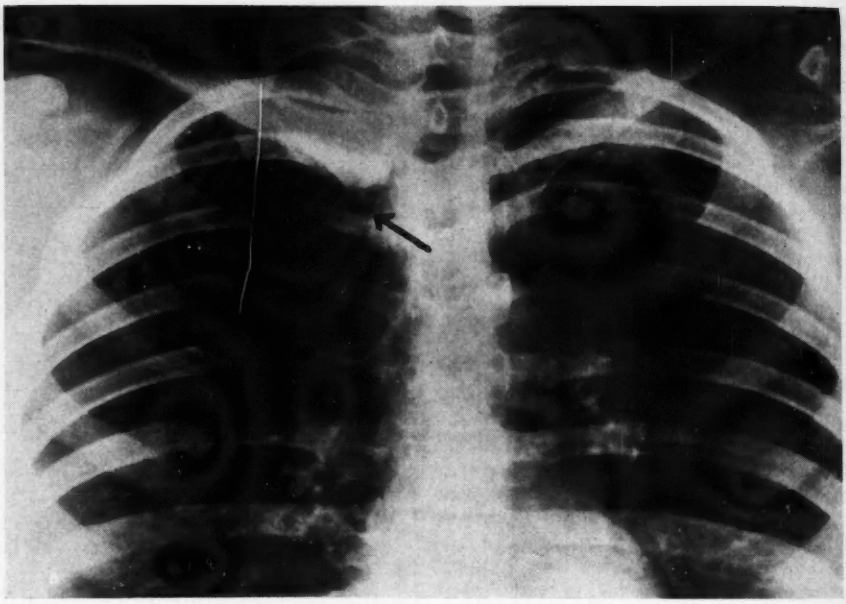


Fig. 8.—Mrs. L.B. Chest radiograph showing a round shadow in right thoracic inlet; antero-posterior view.

cervical portion of the lymphangioma was excised. In so doing the internal jugular vein was deliberately sacrificed and the thoracic duct was doubly ligated. The second operation was performed 12 days later via a left lateral thoracotomy. A semisolid, cystic, lobulated tumour, two inches by two inches in size was removed. It extended down to the hilum of the lung and ramified amongst the branches of the aorta and the innominate vein. This child's chest radiograph two and one-half years later is shown in Fig 11. Clinically the child is cured. This therefore represents a two-pronged attack upon the left thoracic inlet.

Aneurysms of the First Two Intercostal Arteries

CASE 7.—Mr. J.S., aged 49 years (a patient of Dr. A. A. Bailey and Dr. R. B. Lynn), was previously reported upon in 1958.[3] The patient was admitted with a subarachnoid hemorrhage. All neurological examinations including carotid angiograms and myelography were unsuccessful in demonstrating the cause of the hemorrhage. The hemorrhage ceased. A chest radiograph revealed two round calcified lesions overlying the first and second intercostal spaces on the right side (Fig. 12). A diagnosis of aneurysms of the first and second intercostal arteries was made.

At the first operation by Doctor Lynn and the author, the chest was explored. Three aneurysms were found over the necks of the first, second and third ribs on the right side. A large feeding artery descended from above through the thoracic inlet. It could not be safely approached through the chest. Therefore, Doctor Lynn later performed a second operation in which he explored the right thoracic inlet from above and found a large internal jugular vein, 4 cm. in diameter, and a normal common carotid and right subclavian artery. There was, however, a large abnormal artery arising from the innominate artery. This abnormal artery arched up into the neck, then turned down through the thoracic inlet into the thorax to enter the first aneurysm. It was readily ligated. This, therefore, again represented a two-pronged attack on the inlet area.

Aberrant Phrenic Nerve Causing Compression of the Left Subclavian

CASE 8.—Mrs. O.S. was 38 years of age (patient of Dr. J. Mowbray). Fifteen months before the patient was seen, she noted the sudden onset of numbness and blueness of the



Fig. 9.—Mrs. L.B. Lateral radiograph of chest showing round lesion in right thoracic inlet area.

left arm. This lasted 15 minutes and then disappeared. This phenomenon recurred periodically over the next 15 months. It was initiated by such work as washing clothes, washing herself and scrubbing floors. The arm would swell, become tense, blue and painful. The superficial veins were dilated and prominent. The peripheral pulses were normal. A provisional diagnosis of axillary vein thrombosis was made but venograms showed no definite block and a cardiac catheter could be passed readily into the superior vena cava. She was observed for one further year but her symptoms of cyanosis, swelling and pain and paresthesias of ulnar distribution increased. The circumference of the left arm was 2 cm. greater than that of the right arm. The left thoracic inlet was explored and the branch of the fifth cervical nerve to the phrenic nerve was seen to hook around the subclavian vein, partially compressing it. This branch was divided because those from the third and fourth cervical nerves to the phrenic nerve were shown to be adequate for innervation of the diaphragm (Figs. 13 and 14). A fibrous cervical rib was also found and divided and an upper dorsal sympathectomy was performed. Following operation the patient's symptoms were relieved and the swelling in the arm disappeared.

Note.—This is an example of exploration of the thoracic inlet for a vague lesion, which demonstrated a rare anatomical abnormality.

Axillary Artery Aneurysm

Case 9.—Mrs. D.B., aged 29 years, was accidently shot in the right axilla by her three-year-old son. This produced a traumatic aneurysm of the axillary artery. Before the aneurysm could be dealt with the subclavian artery had to be secured in its third part by a supraclavicular approach. The aneurysm was then excised.

Cervical Rib (Osseous or Fibrous) Producing Neurological Symptoms

Six patients with this type of lesion have been treated in the University of Saskatchewan Hospital. Two of these cases will be discussed.

Case 10.—Mr. H.M., aged 49 years, (patient of Dr. J. G. Stratford), had a 20-year history of wasting of the hypothenar eminence and an eight-year history of deformity of the fingers and wasting of the forearm muscles. He had paresthesia of the medial two fingers. In other words, he had a typical ulnar nerve palsy. Radiography showed no suggestion of a cervical rib.

An exploration of the thoracic inlet revealed a classical fibrous cervical rib, over which passed a thickened edematous lower trunk of the brachial plexus. The fibrous band was divided. Six months later there was complete recovery of sensation in the medial half of the hand, and improved function of the forearm muscles.

Case 11.—Mrs. M.C., aged 57 (patient of Dr. J. G. Stratford), was known to have radiological evidence of rudimentary bilateral cervical ribs. Twelve years previously she had had typical symptoms of irritation of the lower trunk of the right brachial plexus with pain, paresthesia and weakness of ulnar nerve



Fig. 10.—Miss M.N. Radiograph of chest showing large shadow in left upper zone.

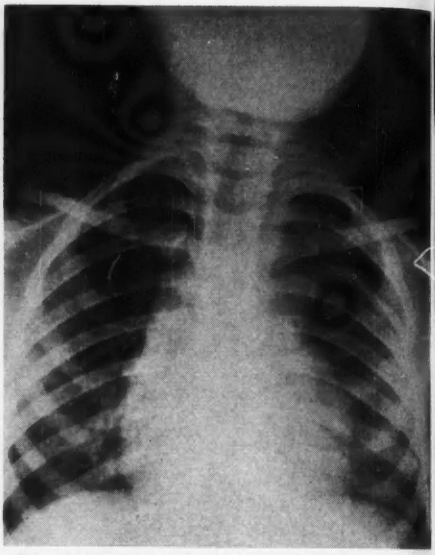


Fig. 11.—Miss M.N. Radiograph of chest taken two and one-half years after removal of extensive cystic hygroma of left side of neck and upper zone of left thoracic cavity.



Fig. 12.—Mr. J.S. Planigram of right upper zone of chest at 9 cm. from the back, showing calcified intercostal aneurysm in second intercostal space.

distribution. At that time she had been explored by a neurosurgeon and had had a scalenotomy with relief. However, over the previous two years symptoms had recurred, and symptoms of lesser severity had developed on the left side.

Therefore, a re-exploration of the right thoracic inlet was undertaken. There had been a re-fusion of the scalenus anticus muscle by fibrous tissue which compressed the lower trunk of the brachial plexus against a fibrous cervical rib which extended up to a rudimentary osseous portion. The scalenus anticus muscle was again divided in the area of the fibrosis. The cervical rib was completely removed and the whole thoracic inlet area was explored. The subclavian artery was freed. This produced complete relief of symptoms. Ten days later the left side was similarly explored and treated with equally good results.

Note.—Some have claimed that scalenectomy is adequate treatment of such cases of cervical rib, a concept with which the author disagrees. The whole thoracic inlet should be explored and freed and the cervical rib should be completely removed. In carrying out this procedure the scalenus anticus muscle is of necessity divided as part of the exploration. There is no difficulty in removing a cervical rib without damage to the cervical plexus, provided that care is exercised. Furthermore, tense fibrous bands are often found in the scalenus medius muscle and these may be the cause of pressure on the lower trunk of the brachial plexus. They should be divided.

Summary and Conclusions

The anterior approach to the upper dorsal sympathetic chain has been described; the value of this approach in exploring the thoracic inlet has been demonstrated in a series of patients with a wide variety of lesions. An article such as this should illustrate certain morals, and advance certain pleas. The morals and pleas of this report are:

1) That surgeons should familiarize themselves with the anatomy of the thoracic inlet.
2) They should master the minutiae of the anterior surgical approach to the cervico-dorsal sympathetic chain, because this approach permits a thorough exploration of the thoracic inlet, with inspection of the subclavian vessels and the brachial plexus.

   This is not possible by the posterior or the axillary approaches.

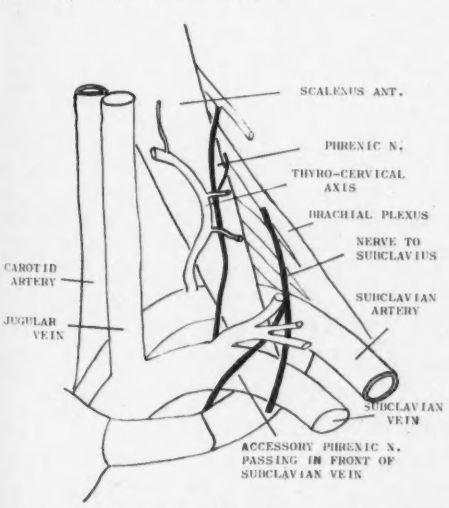


Fig. 13.—Mrs. O.S. Diagram of aberrant root of phrenic nerve causing subclavian vein obstruction.

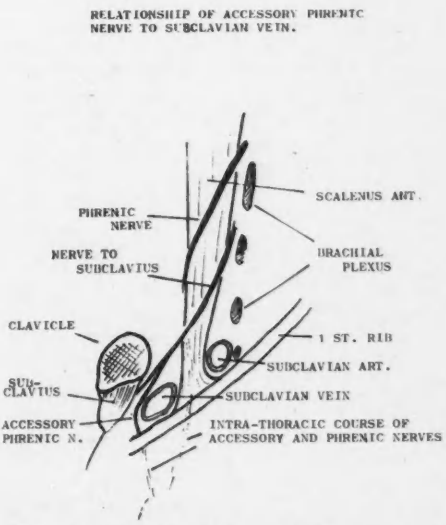


Fig. 14.—Mrs. O.S. Diagram of aberrant root of phrenic nerve as viewed from the side.

3) An exploratory operation should be carried out in patients with neurological and vascular lesions of the upper limb, if there is a reasonable possibility that a causative lesion may be situated in the thoracic inlet area. Such an operation causes no disability to the patient if a negative exploration results and it may pay worthwhile dividends if a lesion is found.

Thoracic inlet exploration should be regarded in much the same light as a laparotomy. In cases of reasonable doubt, don't guess, look!

ACKNOWLEDGMENTS

The author wishes to thank Dr. R. B. Lynn for permission to quote Case 7. He also wishes to thank those physicians and surgeons who referred these interesting problems to him.

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RÉSUMÉ

La cavité thoracique est limitée en haut par l'orifice thoracique supérieur et en bas par le diaphragme. En ce qui concerne ce dernier, il ne présente plus aujourd'hui de problèmes chirurgicaux majeurs. Il n'en est pas de même pour l'orifice thoracique supérieur à cause de son étroitesse: il est limité par les vertèbres thoraciques, la première côte, la clavicule et le sternum; il contient des organes importants: la trachée, l'œsophage, les vaiseaux sous-claviers et carotides, la chaîne sympathique, le premier nerf thoracique, le canal thoracique et le plexus brachial. De nombreuses maladies peuvent nécessiter une action chirurgicale dans cette région: goître plongeant, tumeurs parathyroïdiennes, anévrysmes etc.

L'auteur expose ici les conclusions de ses expériences personnelles: il a eu en effet à intervenir dans l'orifice thoracique supérieur pour les cas suivants: maladie de Raynaud, angine de poitrine, dégénérescence centrale de la rétine, neurofibrome du premier nerf thoracique, syndrome de compression du plexus bracchial par côte cervicale, compression de la veine sous-clavière par un nerf phrénique aberrant, anévrysmes d'artères intercostales (1ère et 2ème), anévrysme de l'artère axillaire.

Il considère que la clef de la bonne compréhension anatomique de cette région est la dissection par une voie d'approche antérieure vers la chaîne sympathique cervico-dorsale. Un certain nombre de précautions particulières doivent être observées pour ce faire; le malade doit être placé correctement sur la table d'opération, la tête surélevée d'environ 10° pour éviter la congestion veineuse, et orientée vers le côté opposé du champ opératoire. L'incision doit être courte (environ trois pouces), passant sur le chef claviculaire du muscle sternocléido-mastoïdien. La veine jugulaire interne sera écartée vers la ligne médiane et l'on repérera alors le muscle scalène et le nerf phrénique. La séparation du scalène donne une vue beaucoup plus commode sur la région.

Dix de ces interventions menées pour différents cas sont décrites en détail.

En terminant, l'auteur insiste sur la nécéssité pour le chirurgien de bien connaître l'anatomie du diaphragme thoracique supérieur. Une exploration de cette région, si elle est raisonnablement indiquée, peut aider énormément le travail diagnostique, et si elle se révèle négative, elle ne nuit nullement au malade. Pas plus en tout cas qu'une laparotomie exploratrice.

NEW BINDING FOR THE JOURNAL

Beginning with this issue of the *Canadian Journal of Surgery* a new type of binding, "Perfect" binding, will be used to enable readers to flatten the journal more easily and to facilitate the removal of pages as desired.

CASE REPORTS

BRONCHOESOPHAGEAL FISTULA ASSOCIATED WITH ESOPHAGEAL DIVERTICULUM

G. E. MILLER, M.D., B.Sc., M.S.,* *Calgary, Alta.*

MALIGNANT DISEASE of either the esophagus or the tracheobronchial tree is the commonest etiological factor in the formation of acquired fistulous communications between these two structures, and, according to Coleman and Bunch,[1] accounts for 50% to 60% of such lesions. Without exception these are hopeless cases. Occurring less frequently, but still in significant numbers, are those bronchoesophageal fistulas due to trauma (28%) and those due to a specific infection such as tuberculosis or lues. Those related to tuberculosis most commonly arise as a result of rupture of a caseous focus into both structures. Broncholithiasis may be incriminated less commonly particularly if, as pointed out by Davis,[2] there is a history of coughing up broncholiths. Luetic fistulas apparently develop as a result of ulceration of gummata.

Fistulas complicating tuberculosis and syphilis have been less common in recent years, owing to the advent of specific antibiotic therapy for the primary disease.

Fistulas associated with esophageal diverticula are of relatively rare occurrence and Stewart *et al.*[3] in 1958, were able to find only 21 such cases in the world literature. It is generally felt that these diverticula are produced by contraction of inflammatory lesions in the vicinity of the esophagus. The development of a fistula between the diverticulum and the tracheobronchial tree is the result of either extension of the original inflammatory process or perforation of an inflamed and ulcerated diverticulum into the adjacent structure.

Fistulas associated with an esophageal diverticulum usually communicate with a primary or secondary bronchus and rarely with the trachea itself. Such bronchoesophageal fistulas occur twice as often on the right side, contrary to what might be expected from the close anatomical relationship between the left main bronchus and the esophagus.

These fistulas are usually under 2 cm. in length, extending from the anterior or anterolateral aspect of the esophagus to the membranous posterior aspect of the bronchus involved. Most commonly they are less than 1 cm. in diameter and occasionally may measure only 1 mm. or 2 mm.

Characteristically, these fistulas are associated with paroxysmal coughing after ingestion of fluid or solid foods. During these paroxysms specimens of the ingested material are coughed up from the tracheobronchial tree. These patients frequently give a history of being able to swallow satisfactorily only in the supine position. As a result of the recurrent soiling of the tracheobronchial tree, either bronchitis or pneumonitis complicates the picture and lung abscesses and bronchiectasis are common in long-standing cases.

Hemorrhage may occur from the fistula itself or from the lung abscesses or bronchiectatic segments.

Diagnosis is based upon the history, roentgenologic studies and endoscopic examination. Physical examination may be of little value except to demonstrate other pulmonary changes where these exist.

CASE REPORT

E.L., a 75-year-old white woman, had a three months' history of paroxysms of choking and coughing on ingestion of liquids and to a lesser extent this also occurred with ingestion of solids. The cough was productive of phlegm, coloured by whatever liquid she had been drinking, and sometimes containing tiny particles of food. The phlegm was never blood-stained. This tendency to cough with alimentation could be avoided by the assumption of a supine position before swallowing. There was no actual dysphagia. Although her appetite remained good, the difficulty in ingestion of food had caused a moderate weight loss of 8 lb. over the three months of her illness.

During the two months before the onset

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of these symptoms, she had experienced two severe bouts of "flu", with temperatures ranging to 102° F., chills and general malaise. This condition had quickly responded to antibiotics and she had no pulmonary complications.

The patient's previous history failed to reveal anything pertinent to her present illness apart from a simple mastectomy performed elsewhere 30 years previously for an undefined tumour.

Physical examination revealed nothing of significance and routine chest radiographs were normal. A series of films taken after ingestion of iodochloral revealed a broncho-esophageal fistula extending from a diverticulum on the anterior wall of the esophagus to the posterior aspect of the left main stem bronchus, 2.5 cm. below the carina (Figs. 1 and 2). The total length of the fistula was about 2 cm (Fig. 3).

Bronchoscopy revealed a slightly roughened, inflamed area, measuring about 0.5 cm. in diameter, in the posterior wall of the left lower lobe bronchus just below the upper lobe take-off. Viewed through a right-angled telescope, a narrow passage could be seen extending posteromedially. Esophagoscopy revealed a diverticulum in the anterolateral wall of the esophagus, 24.5 cm. from the upper gingiva and measuring about 0.5 cm. in diameter. No gross evidence of malignancy was observed on either examination, and biopsies from the area were negative.

Other tests included 1st and 2nd strength tuberculin tests which were interpreted as negative. Blood count erythrocyte sedimentation rates and blood serological test for syphilis were all normal. Bronchograms of the left side of the tracheobronchial tree showed no evidence of bronchiectasis.

On April 14, 1960, a left thoracotomy was performed. The esophagus was mobilized above and below its fistulous communication with the left bronchial tree. The fistula, which measured about 2 cm. in length, was then freed from the surrounding tissues. Surprisingly, little reaction was apparent in the area. Two soft nodes, about 2 cm. in diameter were excised and sent for histological and bacteriological examination. The fistula was then excised and the bronchial and esophageal openings were closed with interrupted 000 silk sutures.

The lymph nodes showed only a mild lym-



Fig. 1.—Esophagus containing Salpix is demonstrated. Salpix has filled the diverticulum and spilled over into the tracheobronchial tree. This is demonstrated in both the right anterior oblique and left anterior oblique positions.



Fig. 2.—The esophagus is shown filled with dye, with the left main stem bronchus and subtending bronchi also outlined with dye. This is placed in the left anterior oblique position.



Fig. 3.—The diverticulum and its bronchial communication are again demonstrated, outlined by a radiopaque medium.

phoid hyperplasia, and bacteriologic examination was entirely negative.

The patient made a complete recovery and when seen four months later had regained the lost 8 lb. She was eating and drinking heartily and was about to depart on a trip to Europe.

Discussion

Traditionally, the findings of a bronchoesophageal fistula in a patient of this woman's age is a source of despair to the surgeon in view of the statistical preponderance of malignant etiology of such lesions.

These statistics, however, should not deter one from a thorough investigation of the case in hand, since if benign, it represents a lesion that is readily amenable to direct surgical correction. Curiously, Coleman and Bunch in 1950, reported only 25 cases, successfully treated by operation. Since then, however, numerous satisfactory results from this approach have been recorded.[4-6]

It should be pointed out that where lung involvement is extensive, simple closure of the fistula is inadequate and operation should include resection of the diseased pulmonary tissue.

Conclusion

The etiology of the diverticulum in the case described in this report remains obscure. In the presence of a negative tuberculin test a tuberculous etiology seems unlikely. Presumably she had had a nontuberculous lymphadenitis at some time in her life, which in subsiding, produced a diverticulum. It could be postulated that ulceration and perforation of the tip of this diverticulum resulted in the formation of the fistula. The relationship of the two severe "colds" earlier in the year in which her symptoms developed, is questionable.

The satisfactory outcome in this case demonstrated that a direct approach is by no means as hazardous as might be considered.

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Résumé

Ceci est la présentation d'une histoire de cas. Il s'agit d'une femme de 75 ans, présentant trois mois avant son hospitalisation un syndrome de toux violente lors de l'ingestion de liquides: les expectorations ainsi provoquées se trouvaient colorées par les liquides avalés et parfois même contenaient de petites particules alimentaires solides, mais ne furent jamais sanglantes. Cette toux à la déglutition pouvait être évitée si la malade se penchait ou se couchait en arrière. Il y avait eu une perte de poids d'environ 8 lb.

A l'examen physique on ne trouva rien de significatif. Les radiographies pulmonaires étaient normales. Cependant une série de films faits après ingestion d'iodochloral révéla l'existence d'une fistule broncho-œsophagienne située entre un petit diverticule de la paroi antérieure de l'œsophage et la partie postérieure antérieure de principale gauche à 2.5 cm. en dessous de la carine. Cette lésion fut retrouvée à la bronchoscopie.

On procéda donc à une thoractomie, à gauche. Le trajet fistuleux fut disséqué et l'on ne trouva dans la région que deux petits ganglions lymphatiques. Après excision, la bronche et l'œsophage furent soigneusement suturés. L'examen anatomopathologique des pièces ne montra qu'une hyperplasie lymphoïde discrète: l'examen bactériologique fut négatif. Les suites post-opératoires furent excellentes.

Dans ce cas, l'étiologie reste obscure.

A HISTORY OF PSYCHIATRY. Jerome M. Schneck, Clinical Associate Professor of Psychiatry, State University of New York College of Medicine, New York City. 196 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.00.

The author, in taking as one of his aims in writing this History of Psychiatry "the enhancing of perspective" has set himself a difficult task. He has increased his difficulties by attempting to cover an immense period of time in only 166 pages of text. In the opinion of the reviewer, Dr. Schneck was unequal to the task. What has been produced is a beautifully bound and printed volume containing capsule comments about a great many prominent psychiatric figures. No attempt is made to account for the bewildering variety of thoughts and practices over these many years, so that the reader, while perhaps gaining facts, gains little in understanding or perspective. One is left with the impression that truth about the nature of man and deviant behaviour began to emerge only with Freud and that prior to him, psychiatric thinkers were variously quaint, muddled, mad, kind or sometimes vicious people. When dealing with the story of mankind's attempts to understand and control his physical environment it is possible to make out a good case for the view that there has been progress, in the sense that this implies improvement or enlightenment. Considering the present unvalidated state of knowledge, the lack of positive evidence for the effectiveness of our methods, it is not easily possible to think of the history of psychiatry as the story of progress. What one can do is to make explicit the basic assumptions that exist about the nature of man and then approach history as an account of the thoughts and practices that particular men have developed from these assumptions. For example, one clear trend in history has followed from the assumption that the essence of man is spirit or mind. From this axiom has come the elaboration that this spirit is capable of beng influenced or shaped in ways that determine the behaviour of man. For some, the influencing force has been a primary spirit or God; for others it has been nature as lawful biological force; for still others, society in the variable sense of collective unconscious or collective sentiments and beliefs. Making this assumption explicit allows one to see the connection between the apparently disparate views of "primitive" thinkers, theologians, sociologists, psychoanalysts, and authors of the Malleus Maleficarum and Jungian psychologists. The basic assumption of medical theory that behaviour depends on the function of brain cells and that deviant behaviour is the result of damage to these cells outlines another historical trend. This allows one to see the connection between the thinking of Hippocrates, Galen, Erhlich, Noguchi, Virchow, Sakel, Moniz and others. Yet another trend appears to follow from humanist sentiments rather than theory. This connects Pinel, Tuke, Dorothea Dix and others. It is regrettable that the author found no such means of bringing coherence to his volume. For this reason it is difficult to recommend the book to its intended readers.

CARCINOMA OF THE STOMACH FOLLOWING GASTRECTOMY OR GASTROENTEROSTOMY FOR BENIGN PEPTIC ULCER

W. H. McCRAE, M.D. and I. B. MACDONALD, M.D., M.S., F.R.C.S.[C], *Toronto*

IN RECENT YEARS there have been increasing numbers of published reports of carcinoma of the stomach occurring in patients who had previously undergone gastroenterostomy or partial gastrectomy for benign peptic ulcer. The incidence of malignancy after either of these surgical procedures has been reported to be small. However, Helsingen and Hillestad[1] have concluded from their observations that patients with gastric resections for benign gastric ulcer had an incidence of subsequently developing carcinoma that was three times as great as the incidence of gastric cancer predicted statistically for the general population of Norway. They also reported that patients who had undergone similar operations for duodenal ulcer did not exhibit this increased incidence of subsequent gastric carcinoma.

Côté, Dockerty and Cain,[2] on the basis of an analysis of gastric cancers recorded over a 50-year period, concluded that the small number of cases in which carcinoma arose in the gastric remnant indicated that gastric carcinoma was not a common complication of the surgical treatment of peptic ulcer.

In 1950, Orringer[3] reviewed 1160 cases of cancer of the stomach from the records of Mount Sinai Hospital, New York, and reported a 0.4% incidence of carcinomas developing later in the gastric remnant. This finding supports the non-statistical studies of Côté *et al.* and is in agreement with the observations of other authors.[4-7]

An interest in this problem was stimulated by the following case encountered recently on the surgical service of the Wellesley Hospital,* Toronto.

CASE REPORT

Miss I.B., aged 58 years, had undergone gastroenterostomy for a benign duodenal ulcer in 1928. She remained well and free of gastric symptoms for the next 26 years. In 1954, routine physical examination revealed a firm mass in the region of the pylorus. A laparotomy was performed and a fibrous, benign pyloric stenosis was discovered. After a pyloroplasty the patient enjoyed a further period of good health, although at times she exhibited an iron-deficiency anemia. In 1959, however, several months of weight loss and a shorter period of recurrent epigastric distress led to her admission to hospital.

Upon examination, the patient was found to have an irregular, hard mid-epigastric mass. The liver and spleen were moderately enlarged, firm and non-tender. Laboratory findings included a hemoglobin of 72%, hematocrit of 37.5% and normal serum proteins and liver function tests. She had no demonstrable free acid in her gastric contents. Laparotomy revealed a non-resectable neoplastic mass along the lesser curvature of the stomach with secondary tumour in the celiac glands, about the pancreas and in both lobes of the liver. Her recovery from the laparotomy was uneventful.

In order to determine the incidence of gastric remnant carcinomas at this centre, the files of the Toronto General Hospital were examined and cases were reviewed of gastric cancer diagnosed over the period 1944 to 1958 inclusive.

In all, 1136 cases were indexed under the diagnosis of carcinoma of the stomach during this 15-year period. Of these, 79 were excluded because the records were inadequate for the purpose of this study. This left 1057 cases for review. It is of some interest that 824 (80%) of these patients underwent operation. About one-half of those subjected to laparotomy (440 cases or 41.6% of the 1057 cases) were found to have resectable tumours.

In only four cases in the entire series was carcinoma of the stomach diagnosed after a previous laparotomy for apparently non-malignant disease. Their principal findings together with those of the aforementioned case are listed in Table I.

In Cases No. 3 and 4, no biopsy material was obtained at the time of the first operation by which to verify the benign nature of the original ulcer. Also, the time that

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TABLE I.—Carcinoma of the Stomach Following Resection or Enteroanastomosis for Benign Peptic Ulcer

Toronto General Hospital 1944 - 1958

		Primary operation				Diagnosis of gastric cancer		
No.	*Sex*	*Age*	*Year*	*Type of operation*	*Diagnosis*	*Year*	*Interval (years)*	*Treatment*
1	M	52	1946	Pyloroplasty	Pyloric stenosis	1949	3	Gastric biopsy Cholecystectomy
2	F	68	1944	Posterior gastro-enterostomy	Duodenal ulcer	1947	3	Laparotomy and biopsy of lesser omentum
3	M	54	1941	Polya gastrectomy	Chronic gastric ulcer	1945	4	Gastro-jejunostomy
4	M	73	1955	Anterior gastro-enterostomy	Duodenal ulcer	1956	1	Laparotomy and enteroenterostomy
5	F	27	1928	Gastroenterostomy	Duodenal ulcer	1959	31	Laparotomy and biopsy

elapsed until the diagnosis of cancer was established, was considerably shorter than that observed by any of those in other centres who have reported their studies of gastric remnant cancers.[1, 7, 8] The time period between incomplete resection of malignant gastric ulcer and the recurrence of malignancy has been reported as approximately one to three years,[9] and such was the elapsed time in three of the five cases listed in this series. If none of these patients had cancer at the time of their first operation, the Toronto General Hospital incidence of gastric remnant carcinomas would be but 0.4% at most.

Summary

A case is reported in which gastric cancer was diagnosed 26 years after gastroenterostomy was performed for duodenal ulcer. Of a total of 1057 well documented cases of gastric cancer diagnosed between 1944 and 1958 at the Toronto General Hospital, four patients had previously undergone operation for supposedly benign peptic ulcer. The incidence of gastric remnant cancer at this hospital for the stated period of 15 years did not exceed 0.4% and was probably less. This incidence is at least as low as that reported by Orringer. The opinion is substantiated that, notwithstanding the statistically increased incidence of this malady in the painstaking Norwegian survey, gastric cancer develops infrequently after partial gastrectomy for benign peptic ulcer.

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Résumé

Sur un total de 1057 cas bien établis de cancer gastrique vus à l'Hôpital Général de Toronto entre 1944 et 1958, quatre patients seulement avaient subi dans leur passé une intervention pour ulcère peptique considéré comme bénin.

Un de ces cas est rapporté ici.

Une femme de 58 ans avait été opérée en 1928 pour un ulcère duodénal bénin: on avait pratiqué une gastro-entérostomie. En 1954 un examen médical de routine permet de découvrir l'existence d'une masse dans la région du pylore: une laparotomie est faite, au cours de laquelle on procède à une pyloroplastie pour sténose bénigne du pylore. Jusqu'en 1959, la malade se porte bien. Peu après, cependant, une perte de poids s'étendant sur plusieurs mois et des troubles gastriques la font réadmettre à l'Hôpital.

On constate alors une tuméfaction intra-abdominale dans la région épigastrique. Le foie et la rate sont légèrement hypertrophiés. L'analyse du suc gastrique indique une absence d'acide libre. Une laparotomie est alors pratiquée: ceci permet de découvrir une grosse tumeur non réséquable située sur la petite courbure gastrique, ayant envahi le pancréas et le foie.

Il semble bien, dans ce cas, que la néoplasie est apparue après les deux premières interventions.

THE CASE REPORTS AND AUTOPSY RECORDS OF AMBROISE PARE. Translated from J. P. Malgaigne's "Œuvres Complètes d'Ambroise Paré", Paris 1840. Compiled and edited by Wallace B. Hamby, M.D., F.A.C.S., Department of Neurological Surgery, Cleveland Clinic, Cleveland, Ohio. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $7.25.

Ambroise Paré is rightly regarded as one of the great figures in surgery and has been called the "father of modern surgery". His life spanned almost the entire 16th century and he was therefore exposed to the ferment of ideas which is termed the Renaissance and which transformed medical philosophy equally with other branches of human thought and endeavour. War too has often been responsible for revolutionizing men's ideas and Paré was the first true military surgeon who learned by his experience and profited from it.

This little book contains his notes and observations on the cases which came under his notice or which he personally treated throughout his career and, though the records are often brief, they are very much to the point and make fascinating reading. Among others we find here his original description of the famous occasion when he applied soothing application in place of boiling oil to gun-shot wounds, his anxiety during the ensuing night as to the outcome and his relief on the following morning when he discovered that the wounded, whom he had treated in this unorthodox fashion, were very much better than those who had received the then orthodox treatment of boiling oil. His comment was "Then I resolved never again to so cruelly burn the poor, wounded by gun-shot".

His well-known dictum "I dressed him, God healed him" occurs frequently in these case records.

This little book is well worthy of being read by all surgeons who are interested in the history of their art, and we are indebted to Dr. Hamby for making it available to us in such a readable translation.

THE SURGEON'S GLOVE. Justine Randers-Pehrson, M.A. 95 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

This is a delightful and well written account of the introduction and acceptance of the use of rubber gloves in surgery. It is obviously based on careful and painstaking research into the literature of the subject. The reader is taken on a guided tour of developments from the earliest hand-covering of sheep's cecum (used to prevent the attendant's fingernails from scratching obstetrical patients) in the middle of the 18th century, to leather, cotton and silk gloves, often used to facilitate surgical manipulation or to protect attendants rather than patients. After these early days, rubber gloves were introduced towards the end of the 19th century and, surprising as it may seem to us, many reputable surgeons were opposed to the use of any type of glove. It is equally surprising to read of controversy over the claims for the superiority of fabric gloves over rubber gloves. During this period it was not always clear whether the gloves were intended to protect the patient or the surgeon. There are interesting sidelights on the rise of antiseptic, and then aseptic methods in surgery, as the authoress shows how the use of rubber gloves became universal as an integral part of the aseptic method.

No biographical details of the authoress can be found in easily available sources, but this slim, well illustrated volume speaks for itself; it is scholarly, interesting and easy to read. There is little to criticize adversely although the distinction between latex-dipped and solution-dipped rubber gloves is not altogether clear, and it is startling to come across a description of the famed Mikulicz as one of the "all-time greats" of surgery.

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SEMINOMA IN A NONAGENARIAN COMPLICATED BY PATHOLOGICAL FRACTURE OF THE HUMERUS

J. G. CONNOLLY, M.D., F.R.C.S.(E)* and E. L. WRATHALL, M.D., *Port Hope, Ont.*

A SEMINOMA is not a common tumour. It accounts for 35%-45% of malignant testicular neoplasms. It is rare before the age of 30, the mean age at operation is in the forties,[1] and it is uncommon in the older age groups. Distant spread is principally by the lymphatics to the regional lymph nodes. Later and less commonly it spreads by the bloodstream to the lungs and liver, and only occasionally involves other organs.

The following case is unusual in several respects and is therefore being recorded.

CASE REPORT

C.M., a 90-year-old man was first seen by one of us (E. L. Wrathall), when he presented with a two-month history of a painless swelling of his right testicle. His other complaints were dyspnea and nocturnal urinary frequency of long duration. There was no history of trauma or instrumentation of the urinary tract.

On physical examination he was found to be a rather well preserved man for his years. Examination of the head and neck revealed no abnormal findings and there were no enlarged cervical lymph nodes. The respiratory and cardiovascular systems were within normal limits for his age. His blood pressure was 155/95 mm. Hg. Abdominal examination revealed no enlargement of the liver, and no abdominal masses were palpable. The right side of the scrotum contained a moderate hydrocele, and the right testicle could be palpated. It was felt to be irregularly enlarged at its lower pole, and the testicular sensation was reduced. The left testicle was found to be atrophic. On rectal examination there was a grade 2 enlargement of the prostate, and the gland was moderately hard. His hemoglobin was 11.8 g. %, the urine contained a trace of albumin but no pus, the blood Wassermann reaction was negative, and the chest radiograph was clear. A tentative diagnosis of malignant testicular tumour with a secondary hydrocele was made.

A skin crease incision was made in the right inguinal region under local anesthesia. The spermatic cord was exposed, ligated at the internal inguinal ring, and the cord and testicle were then removed. Hemostasis was secured, the scrotum was drained and the wound was closed in layers. On opening the specimen, the tunica vaginalis was found to be thickened and the sac contained a straw-coloured fluid. The body of the testicle was enlarged, principally at its lower pole. The tunica albuginea was intact. The cut surface of the testicle was greyish-white in colour, with numerous hemorrhagic areas. The epididymis appeared normal. Microscopic examination revealed a seminoma of the testicle (Figs. 1 and 2).

The patient made an uneventful recovery and was discharged 10 days postoperatively.

The patient was seen next, approximately three years later. At this time he complained of a feeling that "something had given" in his right arm. This had happened that morning



Fig. 1.—Low power view of specimen showing tumour cells infiltrating the tissue (original magnification x 80).

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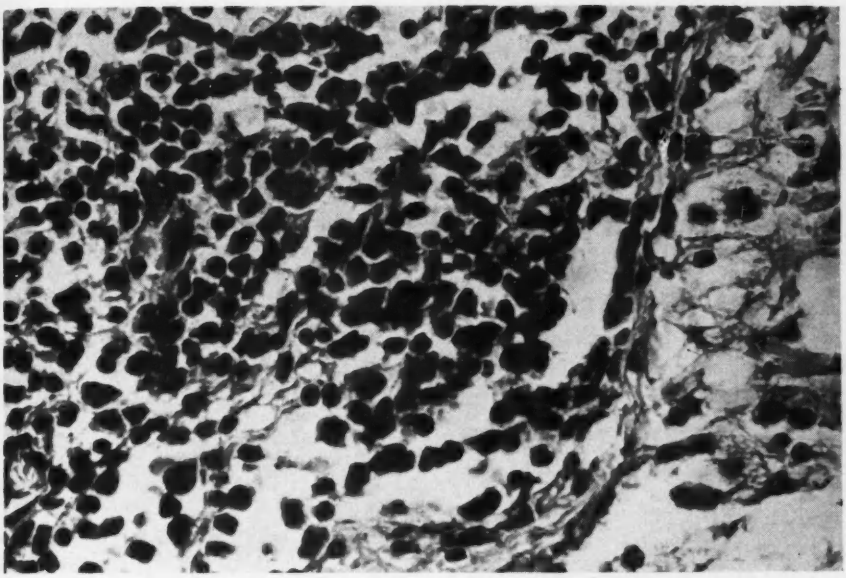


Fig. 2.—High power view of the tumour showing small cells with round nuclei which vary in size and shape and stain very darkly with hematoxylin (original magnification x 500).

while he was opening a drawer. On examination he was found to have an obvious supracondylar fracture of his right humerus. Functional enquiry disclosed no new symptoms since his previous visit. Physical examination revealed a reasonably well nourished old man. There was no cervical lymphadenopathy and the heart and lungs were found to be within normal limits for his age. No abdominal masses were palpable. There was a well healed right inguinal scar. The left testicle was atrophic. The prostatic enlargement was unchanged. His blood pressure was 165/100 mm. Hg, his hemoglobin was 12.2 g. %, and the red blood count was 4.2 million per c.mm. The urine contained 2+ albumin. A radiograph revealed a fracture through a large osteolytic lesion in the lower third of the right humerus (Figs. 3 and 4). A chest radiograph was considered to be within normal limits. A radiological bone survey was carried out and several small osteolytic lesions were noted in the right inferior ischiopubic ramus.

A needle biopsy of the lesion in the right humerus was carried out under local anesthesia. Microscopic examination of the aspirated material revealed a metastatic seminoma (Fig. 5).

Discussion

Testicular tumours represent 2% of malignant tumours in men,[2] and seminoma comprises from 35%-65% of these in the various series reported. Seminomas appear to have a familial incidence and they are the commonest tumours to appear in undescended testes, making up some 80% of these neoplasms.[3]

A simple pathological classification of testicular tumours is difficult. Some pathologists feel that all testicular tumours arise from primitive germ cells and that seminomas are a one-sided development of a teratoma. Others believe that the seminoma is a distinct tumour that arises from the seminiferous tubules. Bell,[4] after a careful study of this subject, was of the opinion that these growths fell into two groups, teratomas and seminomas. Pure seminomas as such do occur but in practically every series one finds evidence of mixed tumours. In one large series, seminomatous tissue was found in 20% of embryonal carcinomas and teratomas.[3] The architecture of testicular tissue is not reproduced in seminomas and the tumour cells do not resemble spermatogonia.[3]

The clinical behaviour of these tumours is variable. Gordon-Taylor[5] divided them into four clinical groups: (1) An "average" type which is slow growing: (2) A "hurri-



Fig. 3.—Radiograph of the lower end of the humerus showing the oblique fracture through the osteolytic lesion in the lower third of the humerus.

cane" type which metastasizes early: (3) An "encapsulated" type which may remain quiescent for years, and (4) "abdominal secondaries", presenting before the primary growth has been discovered. Local symptoms may take the form of a lump in the testicle or of a dragging sensation in this region. The general symptoms may include hemoptysis, dyspnea, abdominal pain, cerebral symptoms, or as in this case, manifestations of a pathological fracture. On local examination there may be a testicular swelling and/or a secondary hydrocele. More extensive examination may reveal abdominal masses, enlargement of the liver or evidence of metastases elsewhere. The differential diagnosis of the testicular swelling includes orchitis, gumma, testicular infarction, tuberculosis of the epididymis, benign tumours, hematocele, simple cysts of the tunica vaginalis, and primary hydrocele.

The pattern of spread of these tumours is well known. Locally they spread to the cord and epididymis and occasionally break through the coverings of the testes to the skin; in such an event the inguinal lymph nodes may be involved. The early distant spread is by way of the lymph vessels which leave the mediastinum testis in company with the veins of the cord and pass to the lumbar nodes. These nodes lie within an area which extends from the renal veins to the level of the bifurcation of the aorta, and from one finger's breadth to the right of the inferior vena cava to one finger's breadth to the left of the aorta. Later, and less frequently, the tumour spreads by the blood stream, principally to the lungs and liver, occasionally to the brain and rarely elsewhere. Badenoch[6] mentions paraplegia secondary to metastatic deposits in the lumbar vertebrae as a presenting symptom in some cases. It is difficult to determine how frequently these tumours metastasize to bone. Unfortunately in some of the largest series such as Friedman's group of 922 cases in service personnel, it was not possible to define the distribution of late metastases.

Summary

A case of seminoma of the testis in a 90-year-old man is presented. Pathological fracture of the humerus at the site of a metastatic deposit occurred three years after removal of the primary growth. The clinical manifestations of this type of neoplasm are briefly reviewed.

Acknowledgment

We wish to thank Dr. J. B. McKay, Provincial Pathologist, for the preparation of the photomicrographs.

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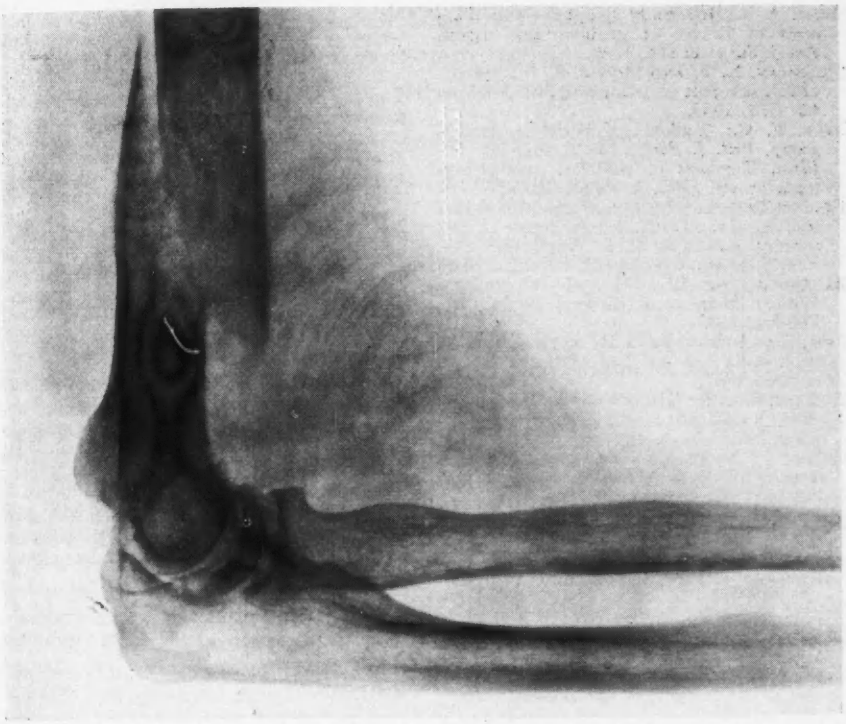


Fig. 4—Lateral view of the lower end of the humerus showing the metastatic deposit and the fracture.

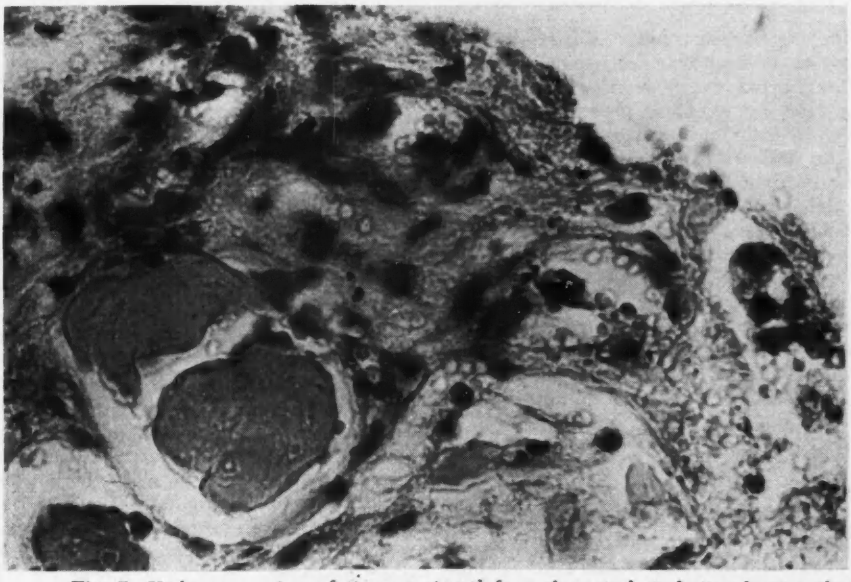


Fig. 5.—High power view of tissue aspirated from the osteolytic lesion showing the typical tumour cells which are darkly staining and vary in size and shape (original magnification x 500).

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RÉSUMÉ

Le séminome est une tumeur rare, ne représentant que 35% à 45% des tumeurs testiculaires. Le cas présenté ici est peu ordinaire à plusieurs points de vue.

Il s'agit d'un homme de 90 ans se plaignant de douleurs et d'enflure de son testicule droit remontant à deux mois auparavant, sans aucun contexte traumatique. L'examen physique ne révéla pas de pathologie générale grave; le scrotum se trouvait distendu du côté droit par un épanchement d'hydrocèle d'importance moyenne, à travers lequel le testicule restait palpable: ce dernier était tuméfié de façon irrégulière à son pôle inférieur; rien à signaler à l'examen rectal, si ce n'est une hypertrophie modérée de la prostate. Les examens de laboratoire étaient normaux. Une exploration fut faite par voie inguinale: le testicule droit fut enlevé de la façon habituelle. Les suites opératoires furent excellentes.

L'examen anatomo-pathologique du testicule montra un séminome.

Le malade fut revu trois ans plus tard pour une fracture de l'humérus droit: à la radiographie, cette fracture était provoquée une large zone d'ostéolyse; des lésions du même genre, mais n'ayant pas entrainé de fractures furent dépistées sur les clichés du squelette. Une ponction exploratrice du foyer huméral ramena des cellules qui étaient des métastases du séminome.

THE PRACTICE OF GRIMNESS

"The genocidal love affair between Americans and automobiles, which resembles nothing in the world so much as a rabbit hypnotized by a cobra, until recently gave no sign of yielding to admonitions or rational thought. Instead, the monster went its way, killing outright nearly 40,000 people a year and at leisure consuming the rest: indispensable, otiose, junky, improbable, it has served as an emotional vehicle for the ungrown and as the backbone of a near-civilization that may be remembered for its superhighways. They do it differently in Erehwon, where the driver of an automobile is quietly executed on the spot, a method that reasonably safeguards him from another accident. In time we too may develop a regard for human life.

"An early sign appeared this summer in Pennsylvania, where all new drivers must have their physician's certification that they can see well enough and have the neuromuscular integrity to control an automobile, and that they are not dyspneic with heart failure, alcoholic, diabetic (uncontrolled), or subject to lapses of consciousness or certain other handicaps. If emotional unfitness is still too hard to assess, though the most lethal of all, the achievement in Pennsylvania is marvelous notwithstanding. It asserts for the first time that driving an automobile is not included in the Bill of Rights. The impression that it was, might have been due to the predecessor vehicle, the horse and buggy, but never extended to, say, flying an airplane.

"Now, and not for the first time, the physician is placed in a pivotal and often distressing role by the law of certification. It will become necessary to exhort these unfit drivers not to drive, and not to *want* to drive, for their own sake. There will be the usual run of highly exceptional circumstances, the routine 'hardships' and special cases, in view of which the physician ought to be 'more reasonable' and the inspired new law edentulous. Really to make the law work, physicians will have to be difficult and even grim. That was the experience in aviation: no one, in order to be a good fellow, signed a poor devil's life away, and the example can well be carried into driver certification. It will be worth any trouble."

Editorial: *Medical Tribune*, Nov. 14, 1960.

EXPERIMENTAL SURGERY

A METHOD OF INTRODUCING BLOOD INTO THE SUBARACHNOID SPACE IN THE REGION OF THE CIRCLE OF WILLIS IN DOGS*

W. M. LOUGHEED, F.R.C.S.[C]† and MARY TOM, M.B.,‡ *Toronto*

INTRODUCTION

THE PURPOSE OF this paper is to report the results of a study of the effects of arterial blood introduced into the subarachnoid space in the region of the circle of Willis. This project was stimulated by the need for better understanding of the pathological and physiological events which occur when a berry aneurysm ruptures. By this method, it is hoped to segregate the effects ensuing after the rupture of an aneurysm, such as arterial spasm and thrombosis, from the effects directly attributable to the release of blood into the subarachnoid space.

Bagley,[1] in 1928, carried out a series of experiments with the injection of blood into the subarachnoid space. However, these experiments differ from those to be described. In Bagley's series, multiple injections were used, and the blood was either introduced into the cisterna magna, or over the cerebral hemispheres. It was felt that better approximation to the clinical course of events might be obtained by injecting blood close to the site at which it arises when an aneurysm ruptures (i.e. the region of the circle of Willis).

METHOD

Adult mongrel dogs weighing between 20 kg. and 30 kg. were chosen for this experiment. Preoperatively, they were brought to the laboratory and allowed to become accustomed to their surroundings. Their actions and responses were carefully recorded. On the day of operation, the animals were anesthetized with intravenous sodium thiopental, and anesthesia was maintained during the operative procedure by an intravenous drip of a solution of sodium thiopental, 1 g. in 500 c.c. of normal saline. When the animal was anesthetized, the spinal fluid pressure was measured either by cisternal puncture or by a lumbar puncture after a laminectomy had been performed.

The needle used for determining the spinal fluid pressure was a No. 20 gauge needle to which was attached a short length of flexible polyethylene tubing (Fig. 1). By using flexible tubing, it could be lowered or raised in order to fill the entire tubing with spinal fluid, and several independent recordings could be made of the spinal fluid pressure.

The procedure of introducing the blood into the subarachnoid space is based on the method of Aschner used for oral hypophysectomies in dogs, as described by Markowitz.[2]

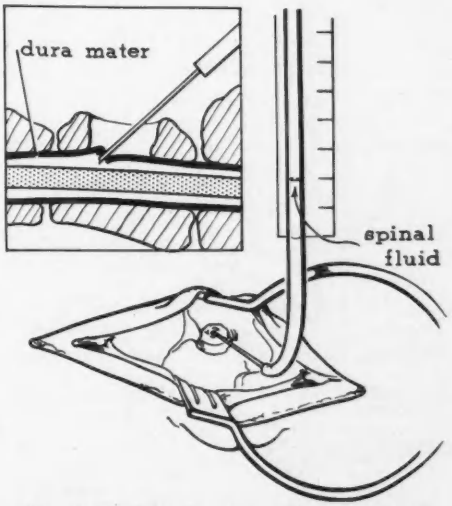


Fig. 1.—Lumbar puncture. The muscles have been reflected off the lamina and the spinous process removed. A burr hole has been made in the lamina to expose the dura. A No. 20 gauge needle is inserted through the laminectomy defect and through the dura into the subarachnoid space. The spinal fluid pressure is measured on a centimetre scale.

*This research has been supported by National Health Grant No. 605-7-162.
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The anesthetized dog is placed on its back and a wide oral exposure is obtained by placing metal rods protected with rubber behind the upper and lower incisor teeth (Fig. 2). The rods are then separated so that the mouth is held open. A suture is placed through the tip of the tongue, which is then tied securely to the neck of the animal so that the tongue is pulled out of the mouth, and over the upper horizontal bar (Fig. 2). The mouth is cleansed thoroughly with tinted tincture of benzalkonium chloride. Sterile towels are used to drape the operative field. The cutting current is used to incise the soft palate in the midline from the point of its junction with the hard palate to within a half inch of the posterior free margin (Fig. 3). The halves of the soft palate are retracted laterally by means of a small self-retaining retractor. The mucous membrane of the nasopharynx is then prepared with benzalkonium chloride, and an incision, using the cutting current, is made in the midline through the mucoperiosteum of the nasopharynx. The mucoperiosteum can then be dissected from the base of the skull by means of a periosteal elevator, and the flaps of mucoperiosteum can be sutured laterally to the retracted edges of the soft palate. In order to obtain better exposure, two small moistened pledgets of cottonoid can be used at the upper and lower margins of the incision in the mucoperiosteum to hold it laterally and also control any bleeding. The exposed bone is washed with saline, and any bony bleeding points are cauterized. The landmark now encountered is the intersphenoid suture between the anterior and posterior sphenoid bones. These occupy the midsagittal plane of the exposure and are separated from the pterygoid bones by two suture lines (Fig. 3). These suture lines, as they progress anteriorly, follow a gentle curving course towards the midline, and at the level of the posterior margin of the hard palate are separated from each other by only one-eighth of an inch. Just caudal to the intersphenoid suture is a small emissary vein which marks the site of the craniopharyngeal canal. Caudal to this emissary vein lies the pituitary gland, and anterior to the emissary vein and intersphenoid suture lies the region of the cisterna chiasmatis (Fig. 3). A small hole can be drilled through the base of the skull just anterior to the emissary vein and intersphenoid suture to expose the dura. A drill, three-eighths of an inch in diameter, has been used for this purpose. Care must be taken to make the drill hole slowly so that the dura is not torn. Bleeding from the bone may be controlled by the use of bone wax. When the dura has been exposed, it should be bluish in colour. This identifies the area as the chiasmatic cistern. If the dura is whitish or pink in colour, the hole has been placed too far posteriorly and lies over the pituitary gland. If this difficulty is en-

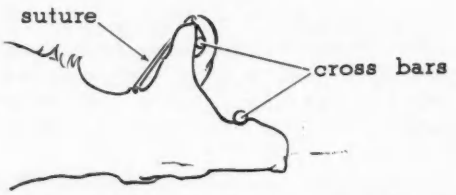


Fig. 2.—The dog is placed in supine position on the operating table. The mouth is held open by parallel rods. The tongue is retracted by suture.

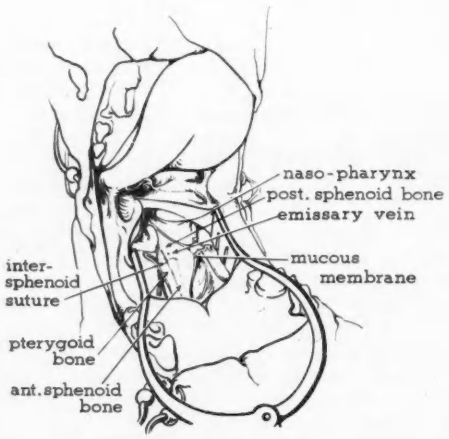


Fig. 3.—The soft palate has been incised and its halves are held retracted. The mucous membrane flaps are held laterally by stitches exposing the base of the skull. The intersphenoid suture line lies in the coronal plane in front of an emissary vein. Two suture lines embrace the sphenoid bones and lie in the sagittal plane gently curving toward each other as they extend anteriorly.

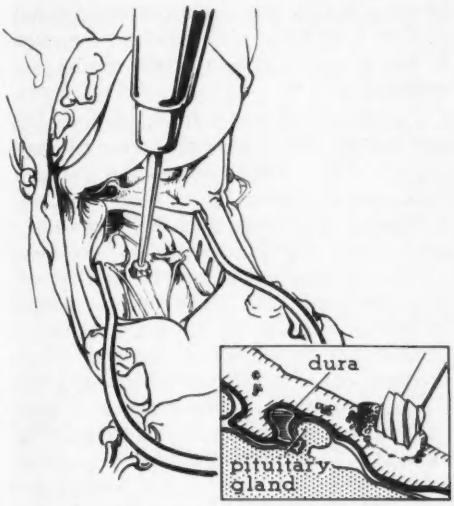


Fig. 4a.—A burr is used to cut an aperture through the bone to the dura just in front of the pituitary gland.

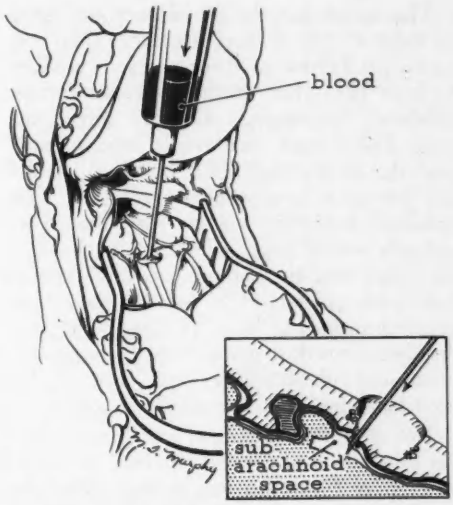


Fig. 4b.—The polythene tubing separating the needle and the syringe is not shown. This tubing is important because it allows the needle to be held still while an assistant outside the operative field carries out the injection. A needle is shown inserted through the dura and arachnoid into the subarachnoid space.

countered, the burr hole must be elongated anteriorly until the bluish-coloured dura has been exposed. The thickness of bone varies from one animal to another, but on the average, it measures one-quarter of an inch. Over the pituitary gland, the bone becomes much thinner, measuring about one-eighth of an inch in thickness. When the dura has been exposed, 5 c.c. of blood is taken from the femoral artery in a siliconed syringe. This syringe is attached to a polythene tube which, in turn, is attached to a No. 21 needle. The needle is then inserted through the dura while an assistant aspirates gently on the syringe (Figs. 4a and b). As soon as spinal fluid is obtained, the blood is injected into the subarachnoid space. When the injection has been completed, the small hole in the bone is filled with gel foam, and sealed by means of a plug of bone wax. The mucoperiosteum is then replaced, and the soft palate is sutured by means of a continuous running suture of plain catgut.

Results

The animals' clinical symptoms were recorded daily and the cerebrospinal fluid pressure was measured preoperatively and before sacrifice. Different animals were autopsied at 24, 48, 96 hours, one week, and two to three months postoperatively.

Fifty experiments were carried to completion. A successful subarachnoid hemorrhage without other existing pathology was achieved in 21 instances (Table I).

TABLE I.—Animals in Which Experiment was Completed

Number of animals in which experiment was completed	50
Successful cases	21
Failures	29

TABLE II.—Causes for Failure

Failures (29)	*Number of animals**	*Clinical symptoms*
Intraventricular hemorrhage	7	Three alive and well, three drowsy, one semi-comatose.
Trauma to third ventricle	8	Three alive and well, five sick.
Meningitis	7	Drowsy
Subdural hematomas	9	All alive and well
Complete failure to introduce any blood into subarachnoid space	4	Alive and well

*These figures add up to more than the total number of failures because some animals had more than one complication.

The causes for the 29 failures are listed in Table II. *Often* there was more than one cause for failure in these cases. It suffices to note here that of the nine cases with subdural hematomas all were alive and well. These cases then would tend to support the theory that 5 c.c. of blood should not act as a space-occupying lesion. The subdural hematomas were encountered in animals which were sacrificed in the first 96 hours and no chronic subdural hematomas were observed. It would appear that in these cases the bevel of the needle had not been inserted completely through the arachnoid membrane so that some of the blood escaped into the subdural space.

Not all of the animals in which a successful injection was carried out had the same amount of blood present in the subarachnoid space. A 1+ hemorrhage was used to designate a spread of blood around the optic nerves, chiasm, pituitary gland and proximal portions of the Sylvian fissures. A 2+ hemorrhage was of the same description but was thicker, with perhaps a thin staining over the brain stem. A 3+ hemorrhage was so classified when the blood spread out on to the hemispheral surface over the temporal lobes and along the base of the brain. The thickness of the blood clot was not used as a basis for grading. A 4+ hemorrhage showed extension into the cisterna ambiens, as well as over the base of the brain and on to the hemispheral surfaces (Figs. 7a and b). No satisfactory explanation was established to account for the variable amounts of subarachnoid blood when the same amount was injected in each case. Some loss may have occurred at the puncture site after the needle was withdrawn.

Among the 21 successful cases there was one death. The remainder survived until the time of sacrifice. Postoperatively, animals varied remarkably in their recuperative power and the clinical findings ranged from ataxia and drowsiness to semicoma (Figs. 5a and b). Ataxia in varying degree occurred after thiopental anesthesia in control animals, so we were unable to evaluate the significance of this sign. When drowsiness and semicoma occurred, it was out of proportion to the amount of anesthetic agent used and was therefore significant. It is noteworthy that the two animals which were semicomatose had a significant elevation of spinal fluid pressure and a 3+ or 4+ hemorrhage. On the other hand 3+ and 4+ hemorrhages were compatible with normal spinal fluid pressure and no signs or symptoms (Table III).

By and large the animals tolerated 5 c.c. of subarachnoid blood well. They tended to be sick for 24 hours but recovered quickly. Six of the 21 showed significant clinical

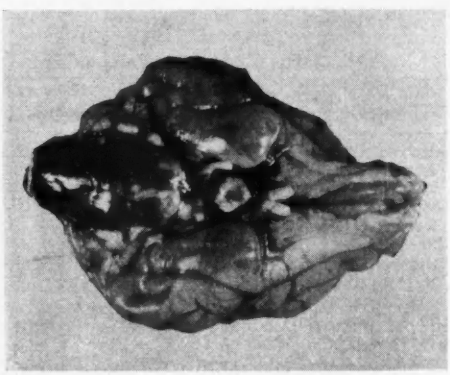


Fig. 5a.—Animal S.3—24 hours postoperatively, alive and well.

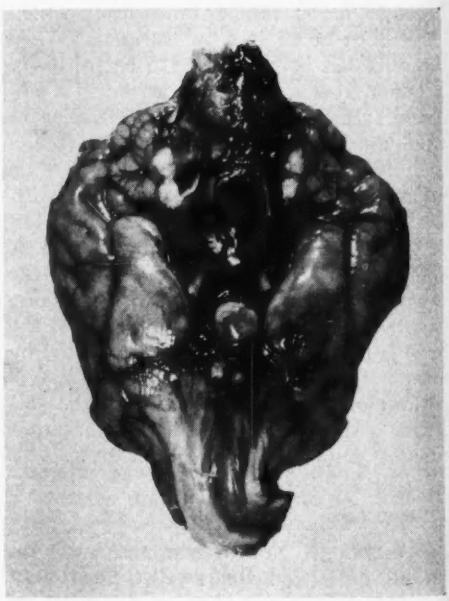


Fig. 5b.—Animal S.9—Comatose, 24 hours after injection.

TABLE III.—21 DOGS WITH SUBARACHNOID BLOOD UNCOMPLICATED BY BRAIN INJURY OR SEPSIS

Hours	Case No.	Size of hemorrhage	C.S.F. pressure		Clinical	
			Preoperatively	Postoperatively		
24	1	2+			Alive and well	
	8	2+	60	170	Drowsy	
	9	3+	60	360	Semicomatose	
	16	3+	95	140	Ataxia	
	46	4+	80	120	Drowsy	
	54	4+	90	170	Alive and well	
	59	4+	90	240	Semicomatose	
	63	3+	60	90	Ataxia	
48	30	4+	73	Died too quickly	Dead	
	44	3+	90	120	Drowsy	
	3	3+	40	100	Alive and well	
72	19	3+	50	60	Alive and well	
	23	3+	120	120	" " "	
96	47	2+	30	80	Alive and well	
Three months	31	Evidence of old blood	N.M.*	110	Alive and well	
	32	"	88	60	" " "	
	33	"	98	110	" " "	Hydrocephalus
	34	"	92	112	" " "	Hydrocephalus
	37	"	84	105	" " "	
	53	"	82	94	" " "	
	57	"	88	92	" " "	

*N.M.—not measured.

symptoms. Only one animal died and autopsy failed to reveal any pathology other than the presence of subarachnoid blood.

Seven animals were sacrificed at two to three months — all of these animals were alive and well at the time. None had convulsions. Their spinal fluid pressures just before autopsy were normal. Two of these animals had hydrocephalus. Brain volumes were carried out according to the method of White[3] and no significant brain swelling was observed.

PATHOLOGY

Method.—At the time of sacrifice, colour photographs were made of the brain which was then weighed, and in 10 instances brain volumes were calculated using White's technique.[3] The brains of the 21 animals in which the experiments were defined as successful because of the uncomplicated introduction of blood into the subarachnoid space were studied, as well as the 29 brains from those in which the experiment was recorded as unsuccessful because of brain injury or the like.

The chief observations recorded on gross examination were the site and extent of subdural and subarachnoid hemorrhage, the presence of intraventricular hemorrhage or dilatation of ventricles and evidence of operative trauma to the brain or pituitary gland. In order to rule out the possibility of minute traumatic lesions close to the site of operation, sections of the pituitary gland and the floor and walls of the third ventricle were examined routinely. Other blocks were taken from the cerebral and cerebellar hemispheres and brain stems to determine the reaction of the leptomeninges to the presence of blood in the subarachnoid space. Stains used included hematoxylin and eosin, cresyl violet and Mallory's connective tissue and phosphotungstic and hematoxylin stains.

PATHOLOGICAL OBSERVATIONS

Gross.—The gross observations proved both interesting and useful and frequently were sufficient to distinguish the successful from the unsuccessful experiments. In seven instances, intraventricular hemorrhages were present and, as seen in Table II, in spite of the fact that hemorrhage was massive, three of the animals showed no clinical signs which would have led one to

suspect its presence (Fig. 2). On the other hand, three of the seven were drowsy and one was semicomatose.

A subdural hematoma was present in nine of the failures; all of these animals were alive and well to that time.

One of the fascinating features was the direction of spread of the blood in the subarachnoid space. It tended to extend from the site of injection around the pituitary stalk into the proximal portions of the Sylvian fissures, backward through the interpeduncular cistern and along the ventral surface of the brain stem. If a 4+ hemorrhage was achieved, the blood spread around the cerebral peduncles between the occipital lobes and cerebellum into the cisterna ambiens. This spread would therefore completely encircle the brain stem and fill this portion of the subarachnoid pathway necessary for the normal circulation of cerebrospinal fluid (Figs. 7a and b). Also in those with a 4+ hemorrhage, the blood would enter the cisterna magna from which, in two cases, it had extended upward through the fourth ventricle and aqueduct as far as the third ventricle. The other interesting feature was the large amount of subarachnoid blood which could be present without causing symptoms (Fig. 8). If the animals were not sacrificed until many weeks after the injection, the only remaining evidence of subarach-

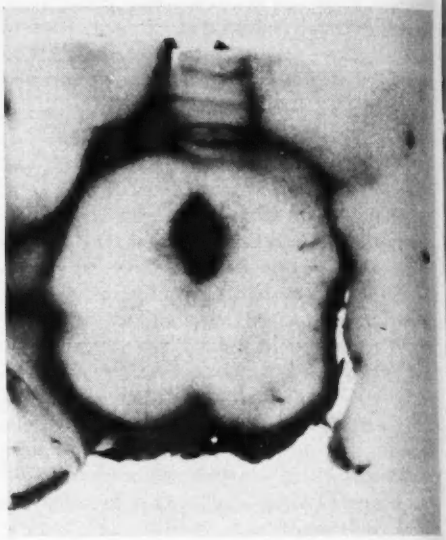


Fig. 7a.—Cross section of dog's brain stem from the interpeduncular fossa to the cisterna ambiens. A collar of blood completely encircles the brain stem.

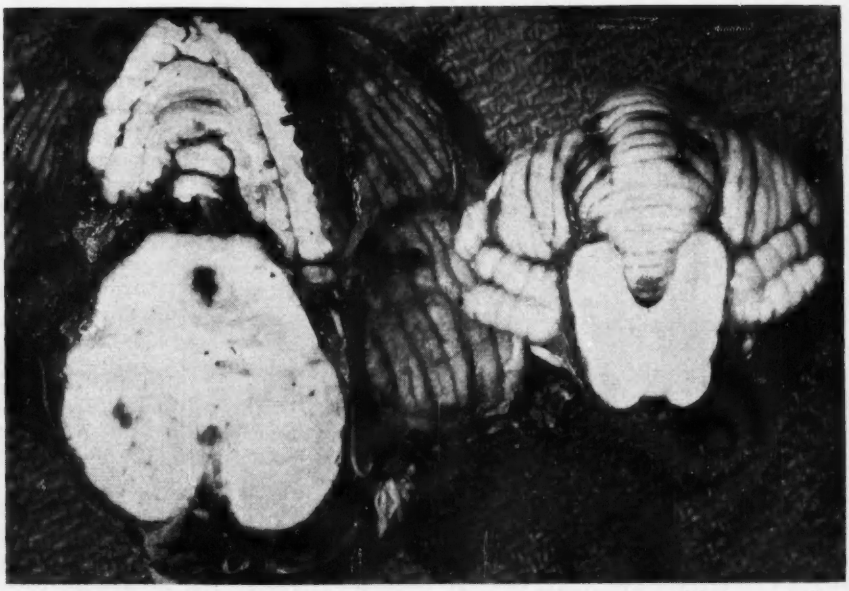


Fig. 7b.—The comparison of a human brain stem with a collar of blood surrounding it on the left, with the dog brain stem on the right.

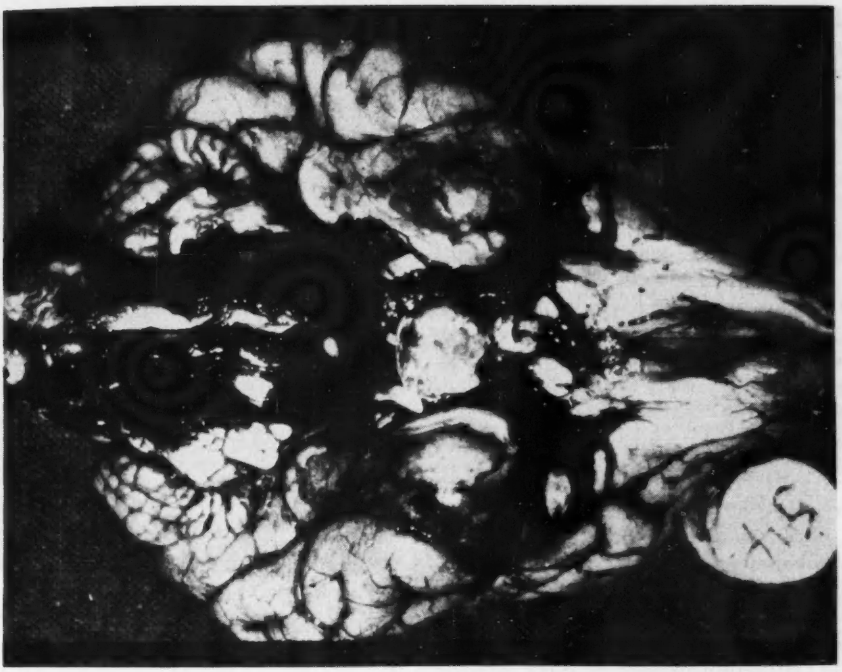


Fig. 8.—A 4+ hemorrhage showing the spread of blood over the base of the brain on to the temporal lobes and around the brain stem. This animal was alive and well despite the extensive subarachnoid hemorrhage.

noid hemorrhage was a faint staining of the brain; there was no evidence of adhesions. In two cases hydrocephalus was present (Fig. 9) but we have encountered this finding in so-called normal animals and do not feel that we have statistical evidence of significance in these experiments.

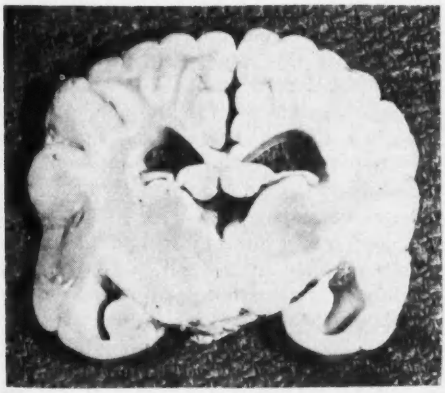


Fig. 9.—This coronal section through the lateral ventricles and tips of the temporal horns shows the extent of hydrocephalus which was present.

Microscopic studies proved very helpful in detecting less obvious failures in the technique. Of the eight cases excluded because of trauma to the region of the third ventricle, three had lesions unsuspected on gross examination. The lamina terminalis was involved in one case and the pituitary gland in two. These lesions were small softenings not associated with intraventricular hemorrhage. Acute meningitis was diagnosed in seven other animals as the polymorphonuclear reaction in the subarachnoid space was much more intense than would be expected if it had been due solely to the presence of blood: these also were excluded.

In the successful cases, microscopic examination showed a moderate polymorphonuclear reaction in the subarachnoid space 24 hours after the injection of the blood. By 72 hours macrophages were present, some of which contained blood pigment

and by the end of a week most of the cells were macrophages. During this period, the fibroblasts in the subarachnoid space showed an increase in size and numbers but this increase was very slight and was not considered to be of any significance. The presence of an occasional hemosiderin-laden macrophage in the leptomeninges accounted for the slight staining of the brain noted in the long term experiments. No significant arachnoid fibrosis was seen in these animals. There was no microscopic evidence of cerebral edema. This finding was supported by the estimations of brain volume.[3] Two small craniopharyngiomas were incidental findings.

Discussion

It would at first seem that 21 successes out of 50 attempts to introduce blood into the subarachnoid space is a discouraging rate. However, in only four of the 50 cases did blood actually fail to enter the subarachnoid space. The failures were occasioned in some instances by minute lesions in the floor of the third ventricle which in several instances caused no clinical symptoms. Other "failures" were so classified because they were complicated by additional, associated lesions.

The survival rate in animals following the uncomplicated introduction of blood into the subarachnoid space is excellent; only one animal died in a series of 21 experiments. The capacity of some animals to tolerate a 4+ subarachnoid hemorrhage or even a gross intraventricular clot without exhibiting any signs or symptoms was indeed surprising (Figs. 6 and 8). However, when the cerebrospinal fluid pressure was significantly elevated, as in cases 9 and 59 (Table III), a 3+ or 4+ hemorrhage was present and the animals were semicomatose. Since none of these animals exhibited a marked cellular response in the leptomeninges to the presence of blood, and since cerebral edema was discovered in no case either by estimation of brain volume or by microscopic studies, it seems fair to assume that the raised cerebrospinal fluid pressure was caused by some factor other than brain swelling. The cause of the increased spinal fluid pressure in these animals has not been established. It is possible that the mode of distribution of the hemorrhage in the subarachnoid space could explain this increase in spinal fluid pressure. We have been impressed by the collar of blood which forms around the brain stem from the interpeduncular fossa ventrally, to the cisterna ambiens dorsally. This occurs at the level of the free margin of the tentorium, and might partially or in some cases completely obstruct cerebrospinal fluid circulation and hence cause elevation of the spinal fluid pressure. This pattern of spread of blood is encountered in patients dying from subarachnoid hemorrhages. Fig. 7b demonstrates the similarity between the human brain with a subarachnoid hemorrhage and the experimental hemorrhage in a dog. This collar of blood might offer an explanation for the onset of acute hydrocephalus which is so frequently encountered in cases of subarachnoid hemorrhage in humans.

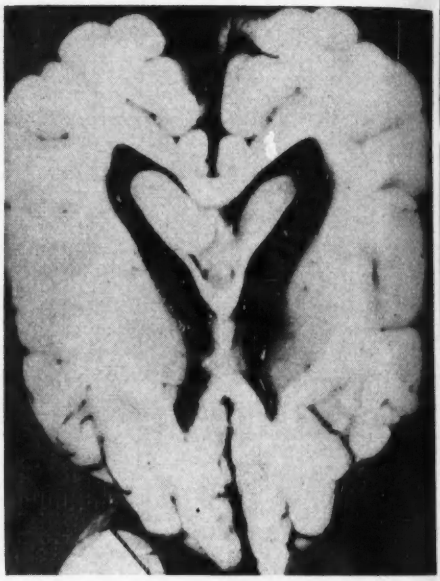


Fig. 6.—This horizontal section through the cerebral hemispheres shows an intraventricular clot, in spite of which the animal was alive and well.

Summary

A method of introducing blood into the subarachnoid space has been described.

The method was successful in 21 of 50 experiments.

A significant rise in cerebrospinal fluid pressure was observed in animals that were semicomatose.

There was no evidence that the presence of subarachnoid blood caused cerebral edema.

It is speculated that spinal fluid pressure changes and clinical symptoms might be explained by acute blockage of the cerebrospinal pathways.

A mechanism for acute blockage of cerebrospinal fluid pathways is suggested from the pathological findings.

ACKNOWLEDGMENTS

The authors wish to thank Dr. E. H. Botterell for his help and advice. We are indebted to Mr. R. Jones and Mr. H. Lane for the photographs taken in the preparation of this paper, and to Miss E. Trites for her help in the preparation of the manuscript.

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RÉSUMÉ

Le présent article se propose d'étudier les effets de l'introduction de sang artériel dans l'espace sous-arachnoïdien dans la région du polygone de Willis, chez le chien. Le but de ces expériences est de mieux comprendre ce qui arrive dans les ruptures anévrysmales.

Ces essais ont été conduits chez des chiens de 20 kg. à 30 kg., dont le comportement était soigneusement étudié avant l'intervention. Après anesthésie au pentothal intraveineux, la pression du liquide céphalo-rachidien fut mesurée. La voie d'accès vers l'espace sous-arachnoïdien choisi fut la voie orale, par laquelle on traverse le palais puis la cavité nasale, au fond et en haut de laquelle on peut alors trépaner la base du crâne; on prépare ensuite une seringue, contenant 5 c.c. de sang, munie d'une aiguille N° 21. On peut de cette façon ponctionner les méninges à travers le trou de trépan. Cette ponction doit être faite avec précaution et lorsque l'on peut aspirer du liquide céphalo-rachidien, on est sûr de se trouver en bonne place: on injecte alors le sang.

Dans ces conditions, on réussit à reproduire une hémorrhagie sous arachnoïdienne dans 21 cas. Les animaux furent étudiés soigneusement quant à leur comportement et quant aux variations de pression de leur liquide rachidien. Ils furent sacrifiés après des périodes variant entre 24 heures et trois mois.

Les auteurs discutent un mécanisme pouvant conduire à un blocage subit de la circulation du liquide rachidien, blocage qui serait susceptible d'expliquer certains symptômes observés.

MAN'S POSTURE: ELECTROMYOGRAPHIC STUDIES. J. Joseph, M.D., M.R.C.O.G. Reader in Anatomy, Guy's Hospital Medical School, London. Introduction by H. Jackson Burrows, M.A., M.D., F.R.C.S. 88 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.00.

This is another splendid book by the Charles C Thomas Publishing House in the monograph series of the American Lectures on Orthopædic Surgery.

Dr. Joseph has chosen to study man's posture within the normal range and has produced a provocative challenge to the common concept of muscle balance in maintenance of posture. The evidence in favour of the role of the ligaments of the knee and the upright posture when the subject is "standing at ease" is very convincing. At the ankle joint, the role of the calf muscles, mainly the soleus, is supported by the study. At the hip joints, the ligaments appear to be the principal factors in stability when the hips are in the extended position.

The author's definition of muscle tone is of particular interest to the clinical teacher. It is suggested that the term should be discarded, or should refer only to the response of skeletal muscle to stretch.

This study with surface electrodes and a suitable amplifier to accommodate the frequency ranges is outlined in detail. The clarity of this presentation makes it a worth-while book in the library of all students of human posture.

The index and bibliography are excellent.

THE ANATOMICAL PATHOLOGY OF EXPERIMENTAL GALLBLADDER CARCINOMA IN HAMSTERS*

G. O. BAIN, M.D.† and K. KOWALEWSKI, Ph.D., M.D.,‡ *Edmonton*

ACCORDING TO Arminski[1] carcinoma of the gallbladder in man is a rare disease having a crude incidence of 0.43% of cadavers. On the other hand, Steiner,[16] in 1942, calculated that any single epithelial cell in the biliary system has a greater chance of becoming cancerous in the life span of the individual than any other cell of the body, except perhaps those of the uterine cervix. Powerful influences must be operative to account for such an exceptional neoplastic propensity. Most discussions of the etiology of cancer of the gallbladder in humans have centred about the high incidence of gallstones in association with cholecystic malignancy. Over the years, many attempts have been made to induce gallbladder carcinoma in experimental animals by the use of gallstones originating either in benign or malignant human gallbladders. In 1933, Burrows[5] reviewed the earlier literature on the effects of placing various foreign bodies in the guinea pig gallbladder and also reported experiments in which gallstones from non-malignant human cases were implanted in the gallbladders of 23 guinea pigs. He interpreted his own results and those of previous workers as having produced only cholecystitis glandularis proliferans. In the same year, Petrov and Krotkina[15] of Leningrad claimed to have produced carcinoma of the gallbladder in guinea pigs by the introduction into the lumen of glass tubes, which in some experiments contained radium. After the second world war, the same authors published further documentation of this experiment.[15] An attempt to reproduce these results in the United States was unsuccessful.[7] The guinea pig gallbladder seems to be exceptionally resistant to the induction of neoplasia. Because of the convertibility of bile acids and cholesterol to methylcholanthrene by chemical processing, the possibility that some chemical carcinogen of phenanthrene type may be secreted in the bile has been considered in the etiology of neoplasia in the biliary tract.[13] We therefore conducted observations on 60 guinea pigs with intracholecystic methylcholanthrene pellets for over 500 days from 1954 to 1956, and found only cholecystitis glandularis proliferans.[19] In the meantime, Fortner[10] produced gallbladder cancer in cats by this technique but, as in the Russian work, the time required was long and the number of animals with tumours was small. We have found the golden hamster a more satisfactory experimental animal for this purpose. In 1958 we reported the occurrence of gallbladder carcinoma in a high proportion of hamsters within eight months following intracholecystic implantation of methylcholanthrene pellets.[3]

The present report deals with new experimental material, confirming and extending our previous observations, with emphasis on the anatomical pathology of experimental carcinoma of the gallbladder.

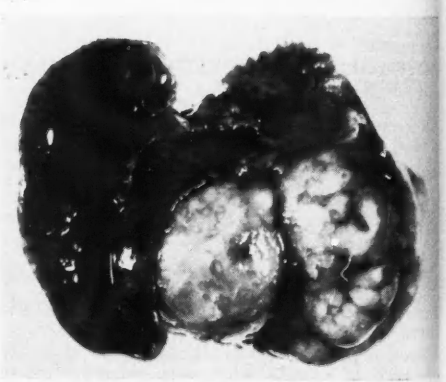


Fig. 1.—Large gallbladder carcinoma which has invaded and replaced part of the liver: 256 days after pellet implanted (Group C).

*This work was supported by Research Grant No. 208 of the National Cancer Institute of Canada. Presented in part at the 49th Annual Meeting of The International Academy of Pathology, Memphis, Tennessee, April 27, 1960.

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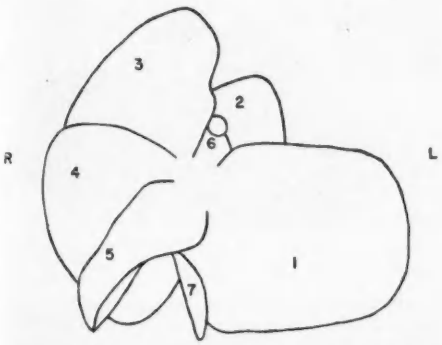


Fig. 2.—Numerals used to identify lobes of hamster liver: caudal surface.

Materials and Methods

Pellets were prepared by melting methylcholanthrene with heat and drawing it into a glass tube of 1 mm. bore. After cooling, the solid methylcholanthrene cylinder was pushed out of the tube and cut into pieces 2 mm. long, each piece weighing from 6 mg. to 8 mg.

The experimental animals were male golden hamsters approximately three months of age, fed on Miracle Rabbit Pellets with water and fresh carrot *ad libitum*. All surgical procedures were carried out under sodium pentobarbital anesthesia (30 mg./kg. body weight). The animals were divided into three groups. The members of group A received single intracholecystic methylcholanthrene pellets implanted by trocar. In group B, the same procedure was carried out, with, in addition, ligation of the cystic duct. In group C, pellet implantation and cystic duct ligation were combined with dissection of the gallbladder from its bed.

Fifty-four of the 59 experimental animals died spontaneously, the other five being sacrificed. All animals were submitted to autopsy examination of thoracic and abdominal viscera. The tissues were fixed in 10% formalin and sectioned in paraffin. All tissues were stained with hematoxylin and eosin and selected sections were also stained by van Gieson, Foot's reticulum and Congo red methods. Certain of the tumours were transplanted by trocar to the cheek pouches of normal hamsters.



Fig. 3.—Carcinoma of gallbladder with residual distended lumen. Tumour has spread to lesser curvature of stomach and mesentery: 288 days after pellet implanted (Group C).

Results

Over 70% of the experimental animals developed carcinoma of the gallbladder within 400 days of implantation of the pellet. In hamsters surviving over 140 days after pellet implantation, the tumour incidence was 77%, and after 240 days it was 81%. There was no significant difference in tumour incidence in the three groups.

Because, in the past, the results of much experimental work have been vitiated through the interpretation of cholecystitis glandularis proliferans as adenocarcinoma, it is essential to discuss the criteria used for the diagnosis of carcinoma in this study. The histological criterion upon which we have placed the greatest reliance is true tumour invasion. In cholecystitis glandularis proliferans, one may observe extensive penetration of pericholecystic tissues by proliferating glandular structures. This does not, however, represent true invasion but rather a herniation of tubular mucosal processes through the muscle. This type of benign glandular invasion therefore represents an extension of the lumen into the gallbladder wall or into surrounding structures. The process rarely extends into contiguous liver and even when it does so, fibrous tissue precedes it. Neoplastic inva-

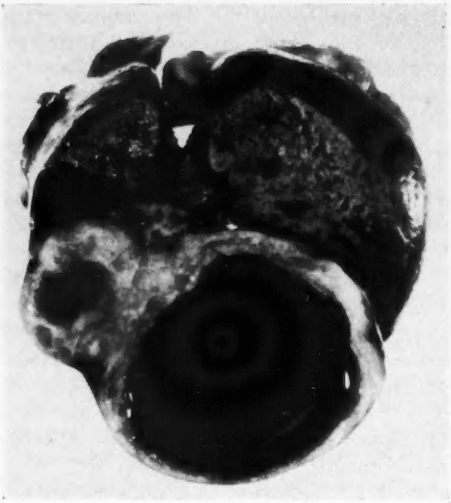


Fig. 4.—Mucocele surrounded by tumour: 256 days after pellet implanted (Group C).

sion, on the other hand, represents a budding-off from the epithelial gallbladder lining and may or may not carry processes of the lumen with it. Although there may be concomitant fibrous proliferation in the gallbladder wall, the neoplastic epithelium is not bound by it but infiltrates through it.

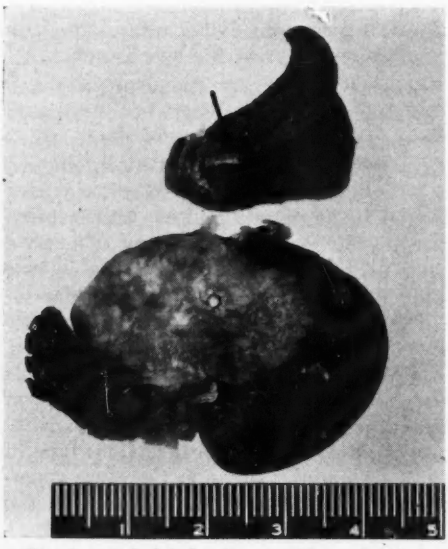


Fig. 5.—Methylcholanthrene pellet embedded in solid tumour. Arrow indicates small subcapsular metastasis: 346 days after pellet implanted (Group C).



Fig. 6.—Gallbladder carcinoma partly surrounding and invading intestine: 327 days after pellet implanted (Group A).

Because of the rigid application of these criteria it is possible that "precancerous" lesions and a few early carcinomas have been excluded. In addition, three non-invasive papillomas of the gallbladder found in groups A and B, 189, 371 and 327 days after pellet implantation have been excluded in calculating the tumour incidence. Two of these (those detected after 371 and 327 days) showed marked cellular hyperchromatism and atypicality in the papiiloma and also the entire gallbladder mucosa.



Fig. 7.—Gallbladder adenocarcinoma invading liver. Distended residual lumen has been emptied. Arrow indicates hepatoma-like nodule seemingly in collision with gallbladder carcinoma: 356 days after pellet implantation (Group C).

Gross Pathology.—The smallest tumours recognized grossly consisted of friable white tissue filling the gallbladder lumen or a solid grey-white mass replacing the organ. In some instances the early tumour formed a thickening or a mass in the wall of the gallbladder. This was seen particularly in animals in which the cystic duct had been ligated, with resulting dilatation of the gallbladder. Larger tumours formed nodular, firm masses bulging from the liver and often replacing much of that organ (Fig. 1). We have identified the lobes of the hamster liver by number (Fig. 2). Lobes 2 and 6 were often entirely replaced by tumour. Lobe 3 was frequently extensively invaded and often replaced. Lobes 1 and 4 were commonly invaded by tumour but even with the largest neoplasms peripheral portions remained. Lobes 5 and 7 were rarely involved. Often the original gallbladder lumen remained either as a cystic bulge on the caudoventral aspect of the tumour or a cyst deep within the tumour filled with green gelatinous or brownish green pultaceous material containing the pellet (Fig. 3). No calculi were found in any of the gallbladders. In animals in which the cystic duct had been ligated the gallbladder lumen was occasionally greatly distended with clear, viscid, mucoid fluid (hydrops or mucocele) as seen in Fig. 4. In other instances no lumen remained, the pellet being incarcerated in tumour tissue (Fig. 5). The cut surfaces of most tumours were grey-white, yellow to green icteric staining being evident in some instances. Necrosis and hemorrhage were common in large tumours and areas of mucoid character were occasionally seen. Many of the larger tumours surrounded and invaded adherent loops of intestine (Fig. 6). In some such instances the stomach and duodenum were greatly dilated at the time of death. One tumour contained a peripheral brownish-pink nodule which resembled hepatocellular carcinoma microscopically (Fig. 7). The remainder of this tumour was pure biliary adenocarcinoma.

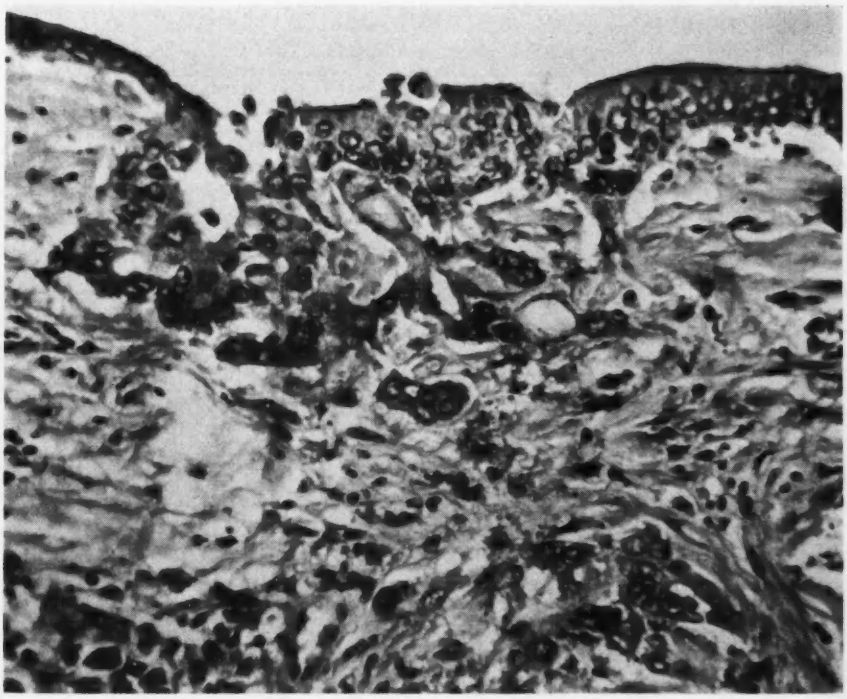


Fig. 8.—Adenocarcinoma of gallbladder. Focus of early invasion of gallbladder wall: 142 days after pellet implanted (Group B; H & E, original magnification x 225).

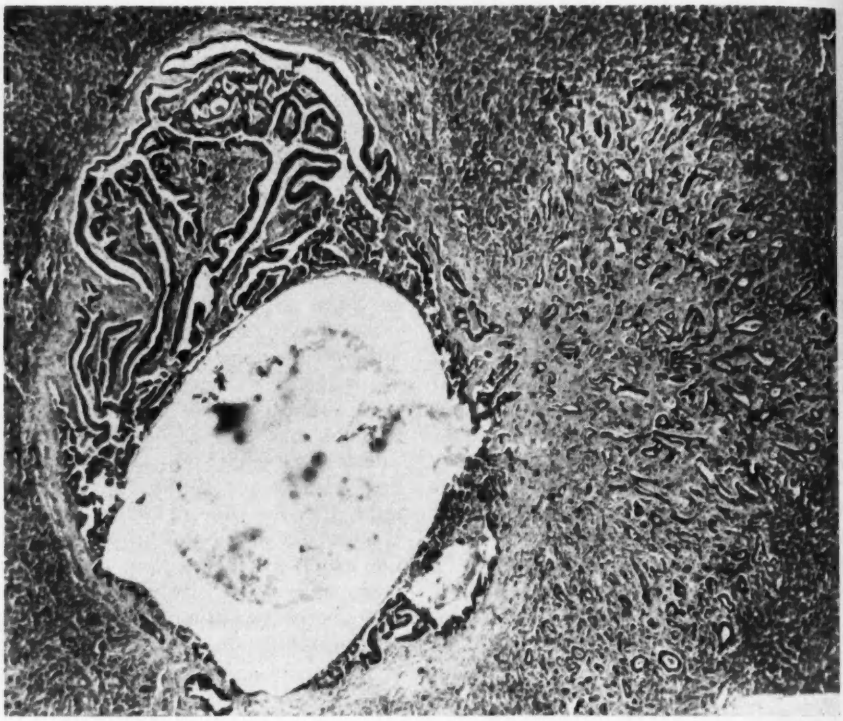


Fig. 9.—Early invasive adenocarcinoma of gallbladder. Lumen occupied by pellet and papillary mucosal proliferation. Tumour invades liver in right-hand side of photograph: 113 days after pellet implanted (Group A; H & E, original magnification x 22.5).

Histopathology.—The earliest epithelial alteration observed in a hamster gallbladder containing a methylcholanthrene pellet was hyperchromatism and anisokaryosis. Sometimes this stage proceeded directly to true invasion of the wall without significant intraluminal growth (Fig. 8). In other instances mucosal epithelial proliferation resulted in pseudostratification and papillary tufting. With further growth, the expanding mucosa was accommodated by complex convolution, resulting in extensive papillary ingrowth and peripheral gland-like outpouching. In this process, the gallbladder architecture was disorganized and the lumen reduced and subdivided. Sometimes the lumen was represented only by the space occupied by the pellet. At the same time, adenocarcinomatous invasion of the adjacent hepatic tissue occurred at one or many points on the circumference of the gallbladder (Fig. 9). In a few instances it was noted in animals in which the cystic duct had not been ligated that the mucosal epithelium of the cystic duct showed proliferation and considerable hyperchromatism and atypia. In one animal the cystic duct was the site of early invasive adenocarcinoma while the lining of the gallbladder proper, although markedly atypical, was noninvasive. It is possible that neoplastic transformation of the extrahepatic bile ducts may have occurred in other animals but was not demonstrable because of incorporation in large tumour masses replacing these structures as well as the gallbladder and much of the liver.

Ninety per cent of the tumours were pure adenocarcinomas, the majority of which were well differentiated (Figs. 10 and 11). Most tumours contained intermediate, small and large glands in varying proportions. In some instances large irregular ductlike structures were prominent (Fig. 12). Papillary formations were common, particularly in association with large gland

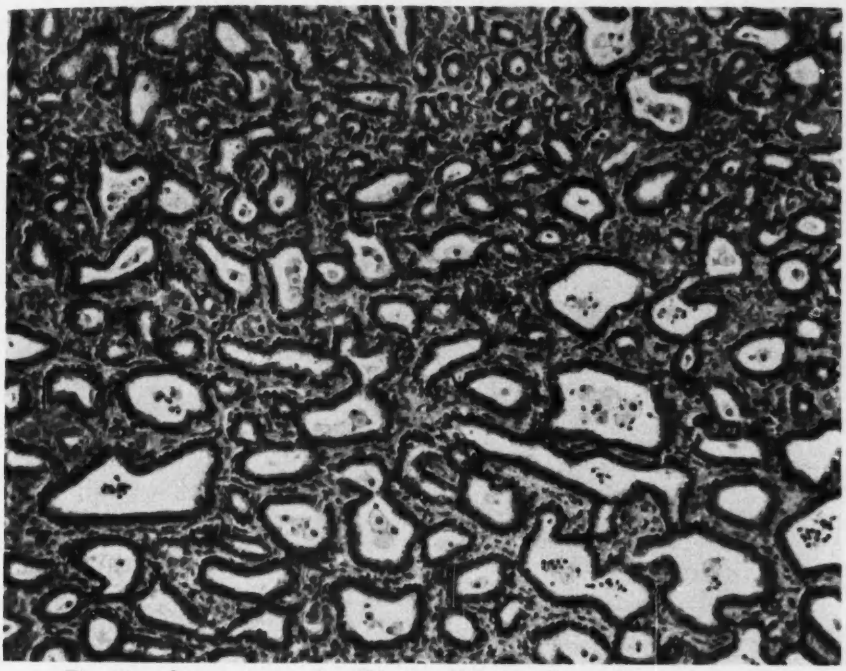


Fig. 10.—Adenocarcinoma of gallbladder: 230 days after pellet implanted (Group C; H & E, original magnification x 90).

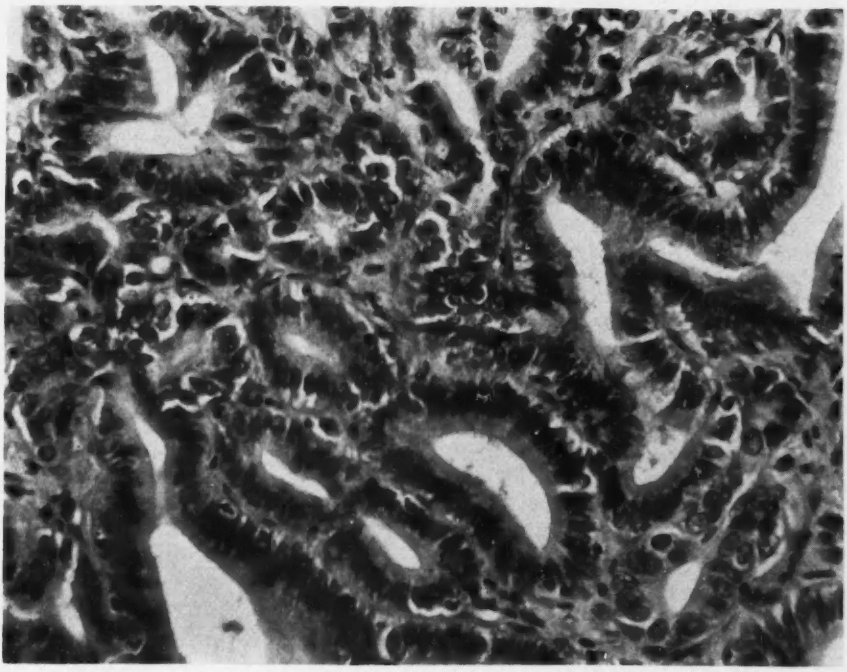


Fig. 11.—Adenocarcinoma of gallbladder: 327 days after pellet implanted (Group A; H & E, original magnification x 225).

or cystic structures. Some tumours contained many mucous goblet cells (Fig. 13). A few of the tumours were poorly differentiated, consisting of sheets of large cells, often with vacuolated cytoplasm, showing little tendency to form glandular or duct structures (Fig. 14). An occasional tumour displayed a sarcomatous type of stroma such as is sometimes seen in experimental hepatoma. In such instances malignant papillary or glandular structures were intimately associated with the sarcomatous type of tumour tissue (Fig. 15). Three tumours were encountered which contained large undifferentiated cells reminiscent of neoplastic liver cells. In one instance the large cells were intimately associated with an adenocarcinomatous component (Fig. 16). In the other two hamsters the large cell components appeared to be in collision with biliary adenocarcinoma of the usual type.

In the hepatic tissue a short distance beyond the invading margins of several of the tumours it was noted that portal bile ducts were lined by hyperchromatic, atypical epithelium. In some instances such ducts were considerably enlarged and apparently empty. Atypical portal bile ducts were rarely seen at any great distance from the tumour. In a few animals examination of the infiltrating tumour margin at its junction with surviving hepatic tissue led to the impression that tumour infiltration, where it met portal triads, progressed slightly in advance of intralobular tumour infiltration. The significance of this observation is not clear as it was not possible to find convincing morphological evidence that tumour infiltration in portal regions progressed by neoplastic transformation of existing portal bile ducts. Nor could this possibility be entirely eliminated.

Both neoplastic and non-neoplastic gallbladders containing methylcholanthrene pellets showed evidence of cholecystitis in the form of lymphocytic and plasma cell infiltration of the wall, often with pus in the lumen. There was commonly loss of muscular tissue with fibrosis and hyaliniza-

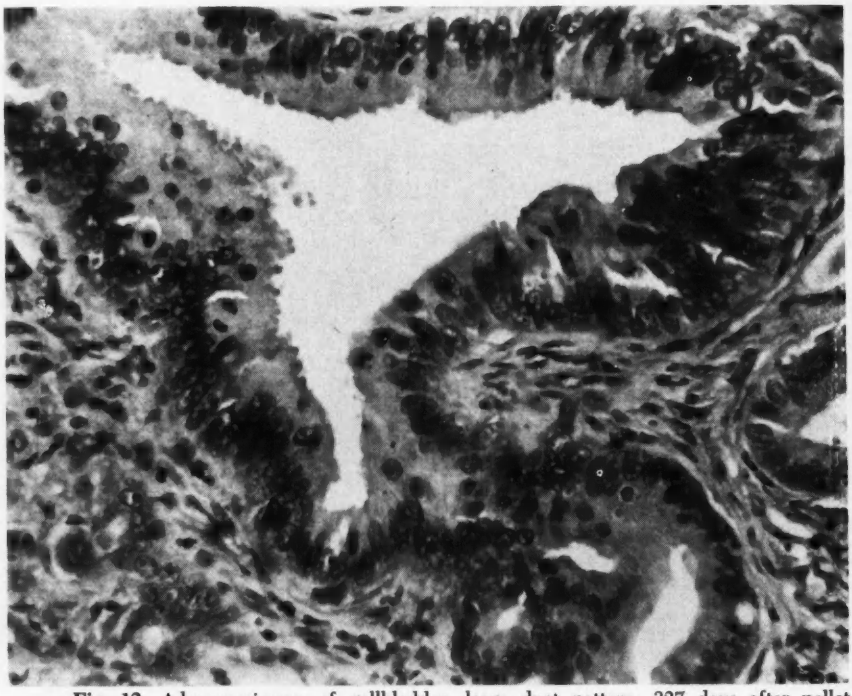


Fig. 12.—Adenocarcinoma of gallbladder, large duct pattern: 327 days after pellet implanted (Group A; H & E, original magnification x 225).



Fig. 13.—Adenocarcinoma of gallbladder. Papillary cystic pattern with mucous goblet cell: 366 days after pellet implanted (Group A; H & E, original magnification x 225).

tion of the wall. In large tumours portions of tumour stroma, especially in the region of the residual gallbladder lumen, often showed similar hyalinization. Extensive necrosis was common in such regions. These inflammatory and degenerative phenomena appear to be a response of mesenchymal tissues to methylcholanthrene. The epithelium of the gallbladder mucosa, although intimately exposed to the methylcholanthrene pellet, did not show this type of reaction but underwent atypical hyperplasia and neoplastic transformation.

In non-neoplastic gallbladders and also in those with small carcinomas there was a proliferation of small bile ducts and occasionally of "oval cells" (*vide infra*) in the gallbladder bed of the liver. Some large tumours showed, in some areas, three more or less distinct zones: a layer of proliferating malignant epithelium lining and projecting into the gallbladder lumen, peripheral to which was hyalinized gallbladder wall containing comparatively little tumour, beyond which was a broad zone of adenocarcinoma invading the liver. This raised a question as to whether the invasive tumour tissue may have arisen from small proliferating bile ducts such as have been observed in the bed of "precancerous" gallbladders and those containing early carcinomas. However, invasion of some portions of the gallbladder wall by malignancy arising in the mucosa could be demonstrated in every tumour in which gallbladder remnants could be identified. Furthermore, study of tumours in various stages of their evolution produced no convincing evidence of malignancy arising in bile duct proliferation in the gallbladder bed.

Tumour Spread.—Vascular invasion was observed in 50% of the tumours (Fig. 17). Excluding hepatic invasion, direct invasion

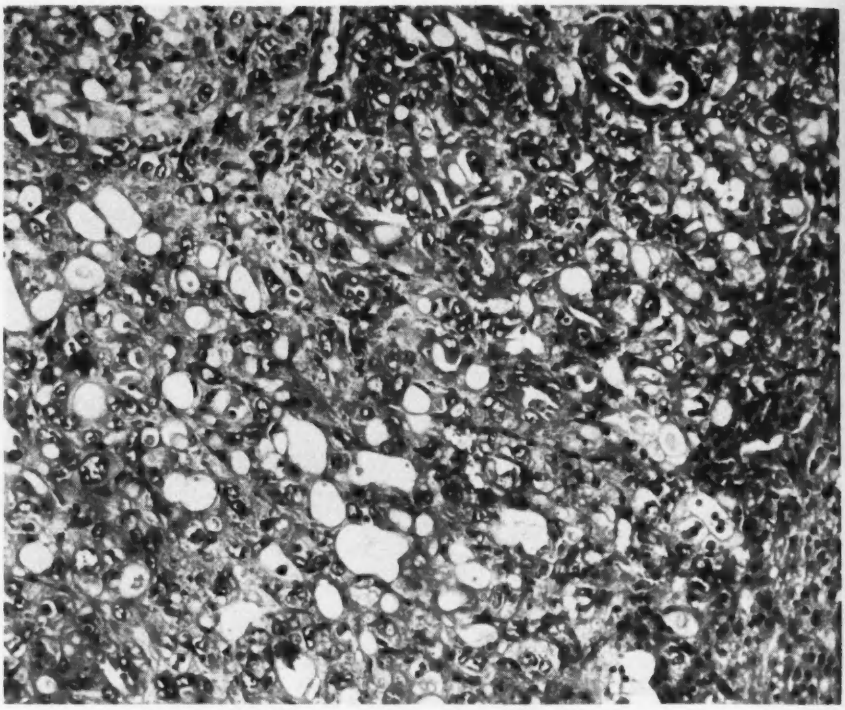


Fig. 14.—Undifferentiated adenocarcinoma of gallbladder invading liver: 168 days after pellet implanted (Group C; H & E, original magnification x 90).

of structures adjacent to the tumour was present in 48%. Structures invaded included the diaphragm (Fig. 18), adherent bowel (Fig. 6), gastro-hepatic ligament (Fig. 19), pancreas and lower thoracic wall. In an occasional animal, intra-abdominal tumour spread was so extensive as to constitute peritoneal carcinomatosis.

Metastases were observed in association with 29% of the tumours. Sites of metastatic tumour included abdominal and mediastinal lymph nodes (Fig. 19), the liver distant from the primary tumour (Fig. 20), the peritoneum, the gastric wall, the diaphragm and the lungs (Fig. 21) and pleura. In a few instances perineural lymphatic metastases were seen. The earliest metastases observed were found in a hamster dying 168 days after pellet implantation.

Transplantation.—One tumour selected at random from each of the three experimental groups was transplanted by trocar into the cheek pouches of normal hamsters. A total of 17 transplants were made. Four of the six transplants from the group A tumour "took"; seven of the eight transplants from the group B tumour "took", and all three transplants from the group C tumour "took". They showed slow but progressive growth requiring about 10 weeks to reach a diameter of 0.5 cm. to 1.0 cm. Some of the transplanted tumours grew as solid nodules (Fig. 22), but others formed rapidly enlarging multicystic structures. Seven of the transplanted tumours have been retransplanted to fresh hamsters with four "takes".

Other Lesions.—A variety of hepatic alterations accompanied many of the tumours of the gallbladder. Hepatic necrosis of variable distribution and degree occurred in 19 animals. Eosinophilic central lobular necrosis was most common, but midzonal, peripheral and focal necroses were also observed. In a few instances massive necrosis of a lobe was clearly associated with invasion and isolation of

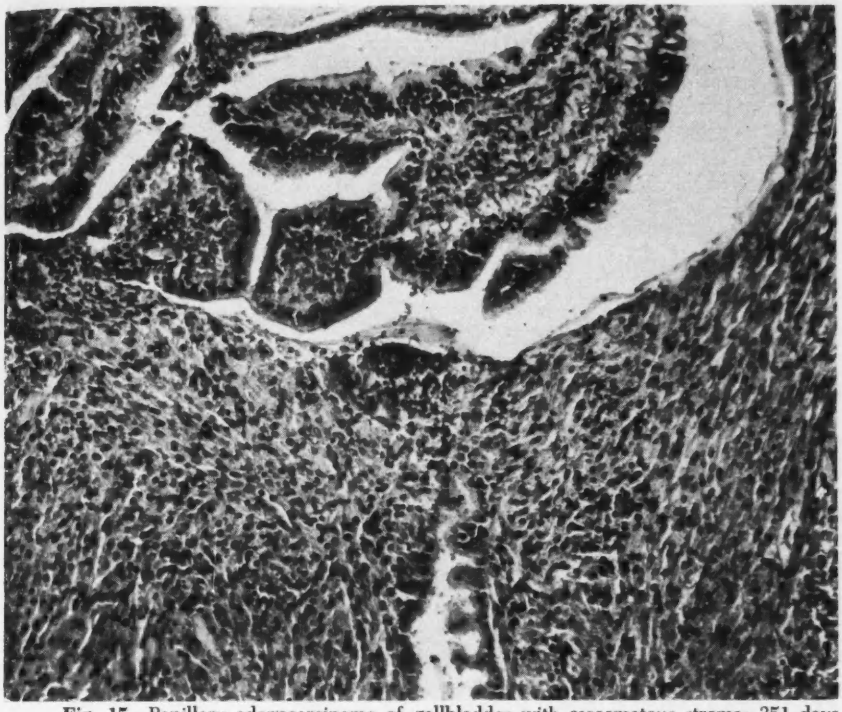


Fig. 15.—Papillary adenocarcinoma of gallbladder with sarcomatous stroma: 351 days after pellet implanted (Group C; H & E, original magnification x 90).

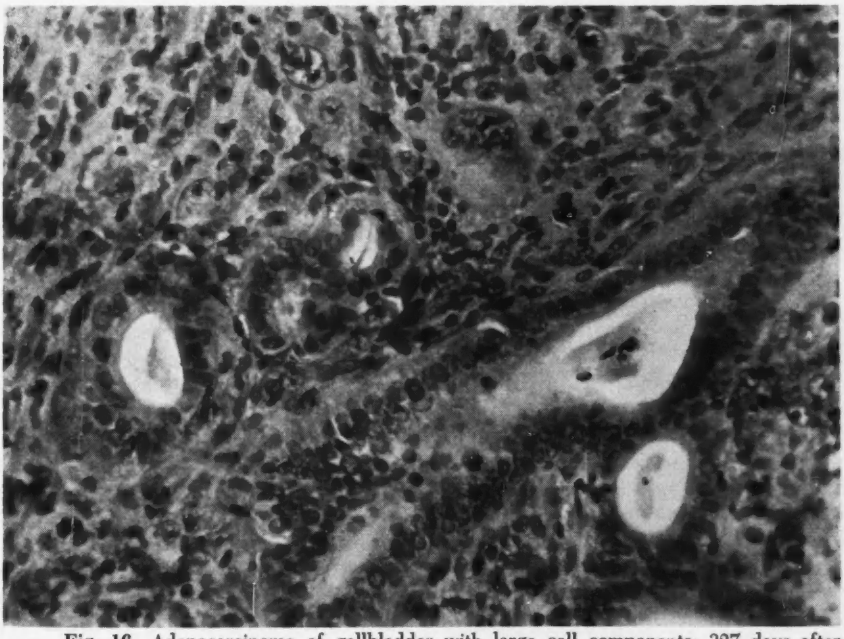


Fig. 16.—Adenocarcinoma of gallbladder with large cell components: 327 days after pellet implanted (Group A, H & E, original magnification x 225).

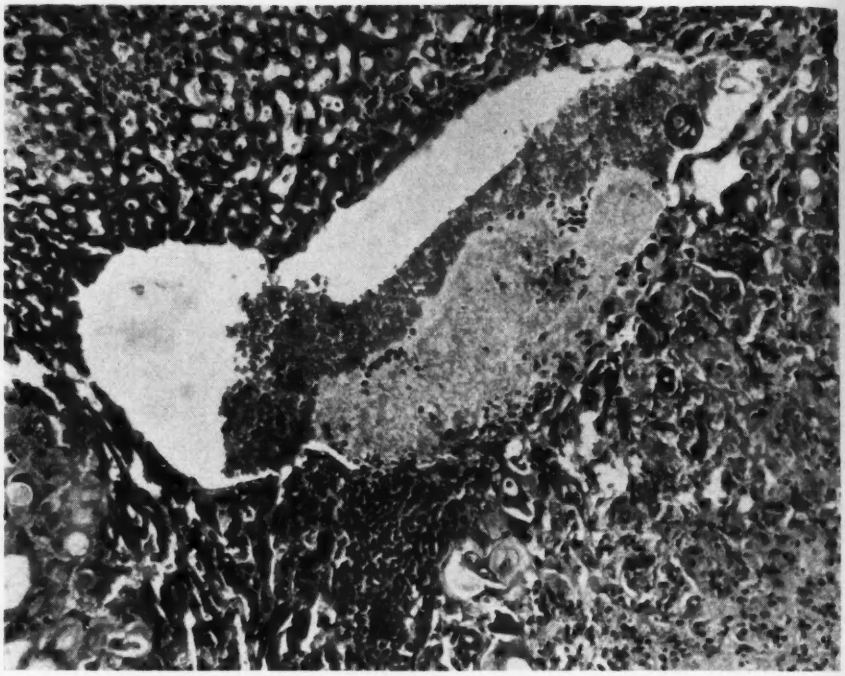


Fig. 17.—Gallbladder carcinoma invading an hepatic vein: 168 days after pellet implanted (Group C; H & E, original magnification x 90).

the lobe by tumour. In many instances necrosis was accompanied by proliferation of pale, oval cells in portal regions (Fig. 23) but the latter also occurred in the absence of hepatic necrosis. In four instances there occurred portal alterations better described as bile duct proliferation with fibrosis. These animals all had gallbladder carcinomas. As in the cholangiofibrosis associated with hepatic carcinogenesis in rats from azo-dye feeding, the newly formed ducts were somewhat atypical (Fig. 24). Twelve livers contained cystic lesions resembling those described in rats fed carcinogenic dyes. In four instances cystic lesions were found in animals without tumours. Most were subcapsular but a few were adjacent to the gallbladder and in one instance the cysts were shown to communicate with the gallbladder lumen. The linings of these cysts are consistent with an origin from biliary epithelium (Fig. 25). In a few instances cystic lesions were contiguous with gallbladder carcinomas but the significance of this is not clear. In several animals mild amyloid deposition was seen in the liver, affecting particularly the subendothelium of hepatic veins. Amyloid was not demonstrated in other organs. The mucosa of the gastrointestinal tract showed no important abnormalities except for the instances in which the gut was the site of direct tumour invasion or metastasis. In a few animals there appeared to be a definite increase in the number of mitotic figures in the mucosal crypts of the small intestine but no primary tumours were seen.

In view of presumptive evidence presented by Dauben and Maybee[6] that C^{14}-labelled methylcholanthrene injected subcutaneously is excreted in the feces, probably by way of the biliary tree, it seemed possible that the various hepatic lesions observed may have resulted from an enterohepatic circulation of methylcholanthrene originating from the pellet.[3] Therefore, in 38 hamsters, after implantation of the pellet, the cystic duct was ligated with or without freezing the gallbladder from its

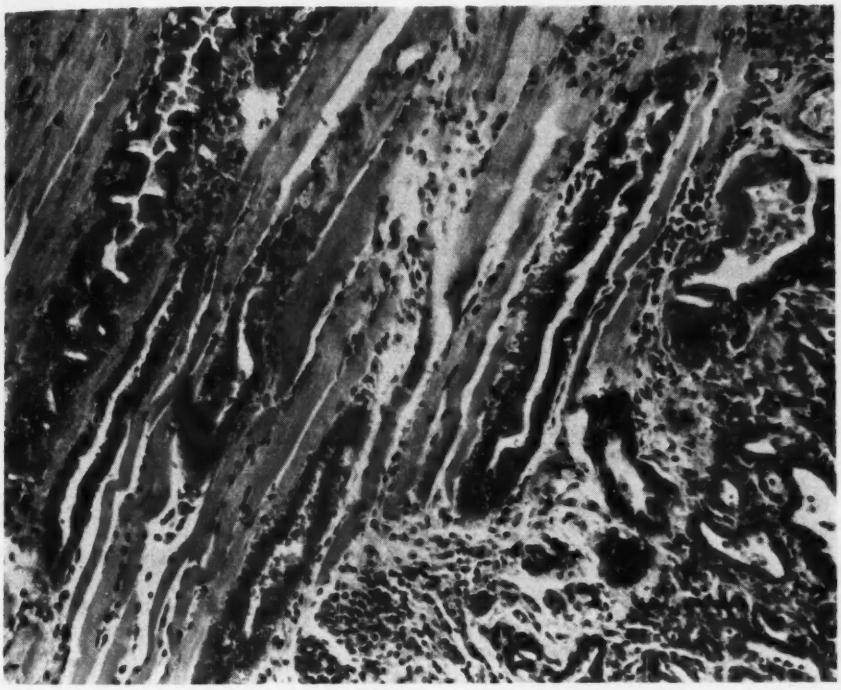


Fig. 18.—Adenocarcinoma of gallbladder invading diaphragm: 230 days after pellet implanted (Group C; H & E, original magnification x 90).

bed (groups B and C). This procedure did not modify the incidence of gallbladder tumours, nor did it affect the occurrence of the hepatic lesions described. Thus, it was not demonstrated that the hepatic lesions are attributable to an enterohepatic circulation of methylcholanthrene. On the other hand, it was observed that portal oval cell proliferation was generally more marked near the gallbladder, diminishing in intensity in more remote portions of the liver. This suggests that the influence stimulating oval cell proliferation may emanate from the gallbladder region. In consideration of the possibility that portal oval cell proliferation may have been caused by bile duct obstruction by enlarging tumour, the livers were examined for evidence of hydro-hepatosis or bile duct dilatation and stasis. Such evidence of biliary obstruction was rarely found; furthermore only an occasional animal had jaundiced body tissues. In addition, when the incidence and severity of oval cell proliferation were compared with tumour size, no correlation



Fig. 19.—Large gallbladder carcinoma replacing part of liver. Nodes at lesser curvature of stomach contain secondary tumour. Gastrohepatic ligament and mesentery invaded: 312 days after pellet implanted (Group A).

TABLE I.—Incidence of Experimental Carcinoma of Hamster Gallbladder

Survival period (days)	*Group*	*Carcinoma*	*Papilloma*	*No tumour*	*Total*
30 - 400	A	14	2	5	21
	B	15	1	5	21
	C	13	0	4	17
Total		42 (71%)	3	14	59
140 - 400	A	13	2	3	18
	B	15	1	3	19
	C	13	0	3	16
Total		41 (77%)	3	9	53

could be demonstrated. Slight oval cell proliferation was found in four animals with no tumours; four hamsters with 1 cm.-4 cm. tumours had no oval cell proliferation; and six animals with 2 cm.-5 cm. tumours had only slight oval cell proliferation. One animal with a tumour only 0.5 cm. in diameter had marked oval cell proliferation.

Discussion

True controls were not possible in this experiment. We are not persuaded that different experimental groups with a variety of intracholecystic foreign bodies as described in some reports represent controls. We are able to state, however, that autopsies on over 250 golden hamsters in this laboratory during the past two years, revealed no instance of spontaneous tumour of the liver or biliary system. Likewise Ashbel[2] found no liver tumours in 1000 autopsies on hamsters.

Ewing[8] found one of the most interesting aspects of gallbladder carcinoma in man to be the high frequency with which it was accompanied by cholelithiasis. The frequency of stones has been variously estimated to be between 70% and 90% of the cases.[1] Zeppa and Womack[18] invoked the hypothesis of carcinogenesis as a two-stage process to explain the part played by stones in gallbladder carcinogenesis. Fortner,[12] pursuing this concept, suggested that the initiating factor was some unknown substance in bile and the promoting factor was the almost invariably present chronic cholecystitis. In the majority of cases the chronic cholecystitis was attributable to stones while in 10% to 30% chronic cholecystitis was of other etiology. Thus, the relationship of stones to carcinogenesis became an indirect one, mediated by chronic cholecystitis. In the present experimental study, inflammatory changes were always present in gallbladders which developed carcinoma and also in those which did not. The inflammatory changes seemed to be attributable to the presence of the methylcholanthrene pellet in the gallbladder lu-

TABLE II.—Invasion and Metastasis of Experimental Carcinoma of Hamster Gallbladder

Survival period (days)	*Group*	*Hamsters with carcinoma*	*Hamsters with direct spread to structures other than liver*	*Hamsters with vascular invasion*	*Hamsters with metastasis*
30 - 400	A	14	8	7	1
	B	15	4	3	4
	C	13	8	11	7
Total		42	20 (48%)	21 (50%)	12 (29%)
140 - 400	A	13	8	6	1
	B	15	4	3	4
	C	13	8	11	7
Total		41	20 (49%)	20 (49%)	12 (29%)
240 - 400	A	10	4	6	1
	B	6	1	2	1
	C	9	4	7	6
Total		25	9 (36%)	15 (60%)	8 (32%)

men. Under the experimental conditions there would seem to be no reason to impute cocarcinogenic activity to cholecystitis since methylcholanthrene is a complete carcinogen possessing both initiating and promoting activity.

Although our data does not permit satisfactory statistical comparison in this regard, examination of the results suggests that surgical manipulation at the time of pellet implantation may have had some influence on the rate of tumour growth and the incidence of metastasis. Those animals in which the cystic duct was ligated developed tumours of larger average size and a higher incidence of metastasis than did those with only pellet implantation. Animals with cystic duct ligation and freeing of the gallbladder from its bed had the largest average tumour size, the highest incidence of vascular invasion and the highest incidence of metastasis.

In our work the occurrence of oval cell proliferation, cystic lesions and bile duct proliferation with fibrosis remains unexplained. Such evidence as can be drawn from the study is of a negative nature, suggesting that mechanical factors are not to be implicated. In this connection Fortner's finding of cystic lesions and cirrhotic changes in the livers of two hamsters dying after 15 to 16 months of repeated subcutaneous injections of bile from human patients with cancer of the common bile duct is of considerable interest.[9] Each of these two animals also had an hepatic carcinoma of bile duct type. In a subsequent publication Fortner[11] interpreted as precancerous some of the intrahepatic bile duct changes in hamsters treated with human bile from cases of extrahepatic biliary tract cancer. The similarity of both the tumour type and the non-neoplastic lesions in Fortner's hamsters to our own material is remarkable and lends some support to the concept that these lesions may be attributable to a chemical carcinogen or some metabolic derivative thereof. Oval cell proliferation, cystic lesions and bile duct proliferation with fibrosis are well known in the field of experimental hepatic carcinogenesis.[17] Opie,[14] studying the pa-

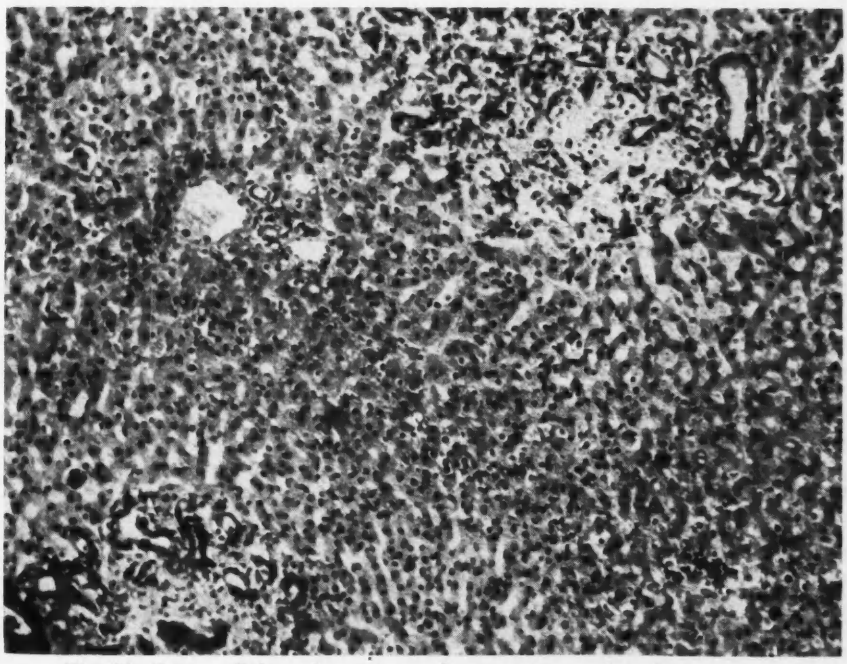


Fig. 20.—Two small foci of metastatic adenocarcinoma in liver from primary adenocarcinoma of gallbladder: 258 days after pellet implanted (Group C; H & E, original magnification x 90).

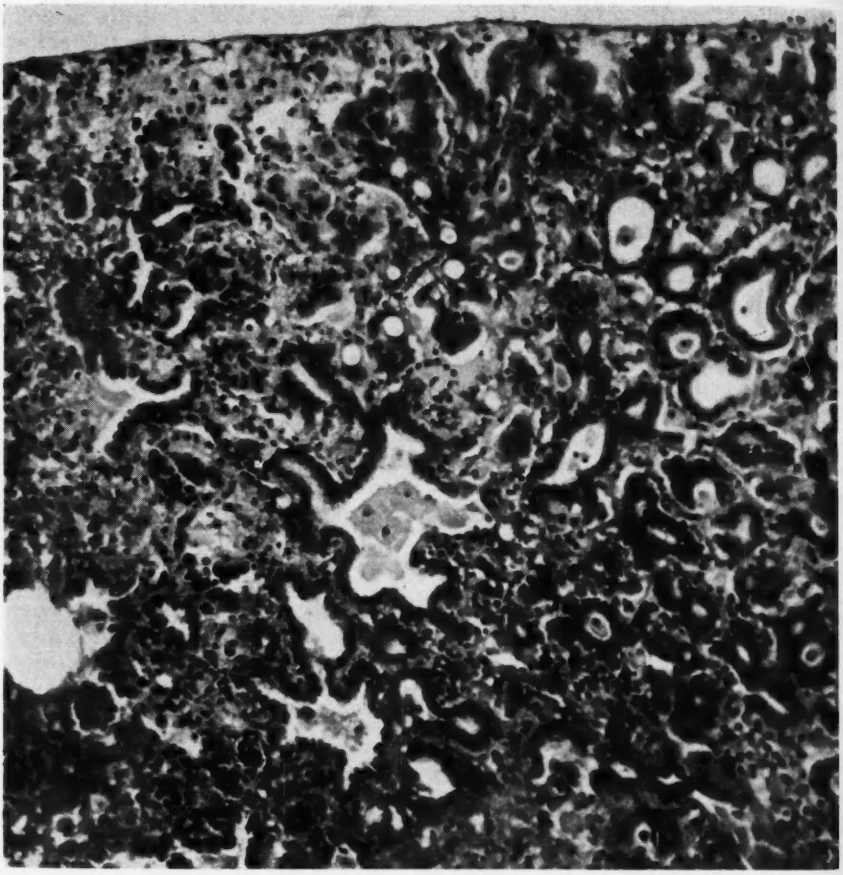


Fig. 21.—Metastatic adenocarcinoma in lung from primary adenocarcinoma of gallbladder. Pleural surface at top of photograph: 346 days after pellet implanted (Group C; H & E, original magnification x 90).

thogenesis of hepatic tumours produced in rats by butter yellow, came to the conclusion that cystic lesions and cholangiofibrosis are preneoplastic lesions. We have not been able to find convincing evidence of the neoplastic transformation of similar lesions observed in our experimental material.

The marked difference in the susceptibility of guinea pig and hamster gallbladders to carcinogenesis under identical conditions remains unexplained. It is startling that the epithelium of the guinea pig gallbladder should remain immune to the carcinogenic influence of methylcholanthrene, one of the most potent carcinogens known, in spite of prolonged and intimate contact.

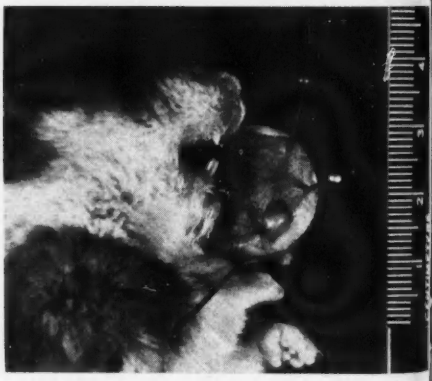


Fig. 22.—Hamster cheek pouch showing first generation tumour transplant from an experimental gallbladder carcinoma.

Even among hamsters, different animals vary greatly in susceptibility to carcinogenesis by methylcholanthrene, for about 25% failed to develop carcinoma in spite of experimental periods of 30 to 351 days and among animals with neoplasm, rates of tumour induction and growth varied widely. Thus, one hamster had a large tumour with distant metastases within 168 days while others had small, only locally invasive tumours as long as 360 days after pellet implantation. Even in the experimental animal exposed to powerful inducing and promoting factors, some individual susceptibility factor plays a major part in carcinogenesis. The fact that the experimental animals were random-bred may be of importance in connection with this variation. However, Berenblum[4] has commented that a wide variation is observed in the latent period of carcinogenesis even among animals (mice) of isogenic strain. In spontaneously occurring gallbladder cancer in man, individual susceptibility undoubtedly also is of great importance and may play a major part in obscuring etiological and pathogenic factors in epidemiological studies of human gallbladder carcinoma. It may be for this reason that clinical and pathological studies of human carcinoma of the gallbladder have yielded so little definitive information on the etiology and pathogenesis of this disease. It may be that the use of an experimental model by which gallbladder carcinoma can be regularly produced by a standard technique will present opportunities for the elucidation of factors which may influence the induction, growth rate and metastasis of carcinoma in this organ.

Summary

Experimental carcinoma of the gallbladder has been produced in over 70% of 59 golden hamsters following the intracholecystic implantation of methylcholanthrene pellets. The malignant neoplastic nature of the tumours has been confirmed by the demonstration of destructive invasion of normal tissues, vascular invasion, lymphatic

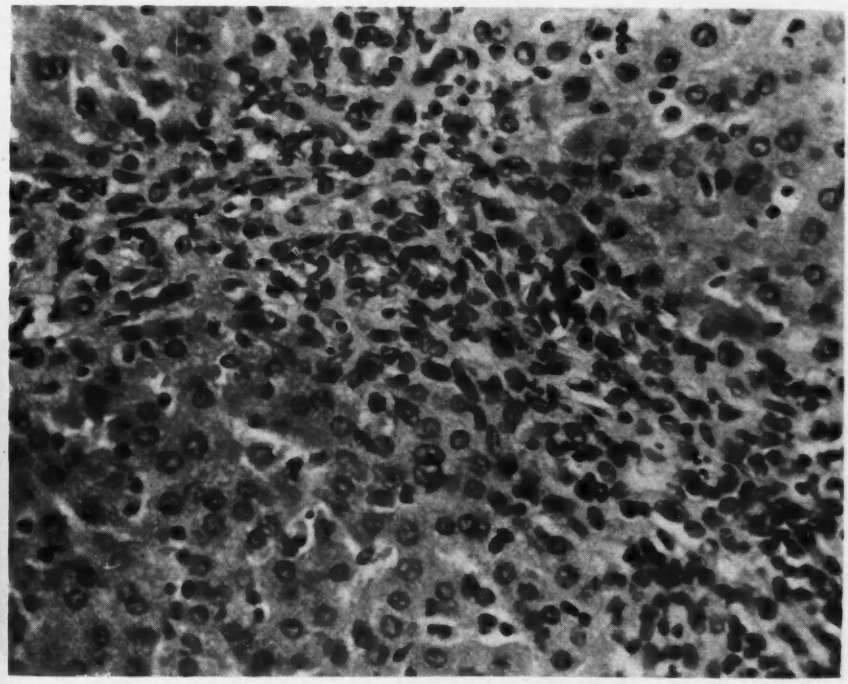


Fig. 23.—Oval cell proliferation in hamster liver: 334 days after pellet implanted (Group C; H & E, original magnification x 225).

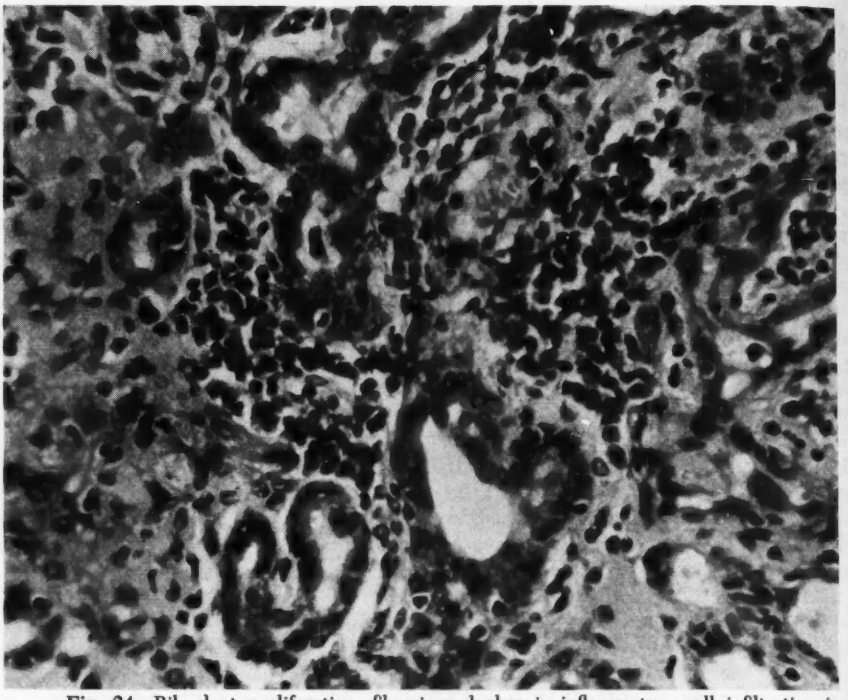


Fig. 24.—Bile duct proliferation, fibrosis and chronic inflammatory cell infiltration in portal region of hamster liver: 214 days after implanted (Group B; H & E, original magnification x 225).

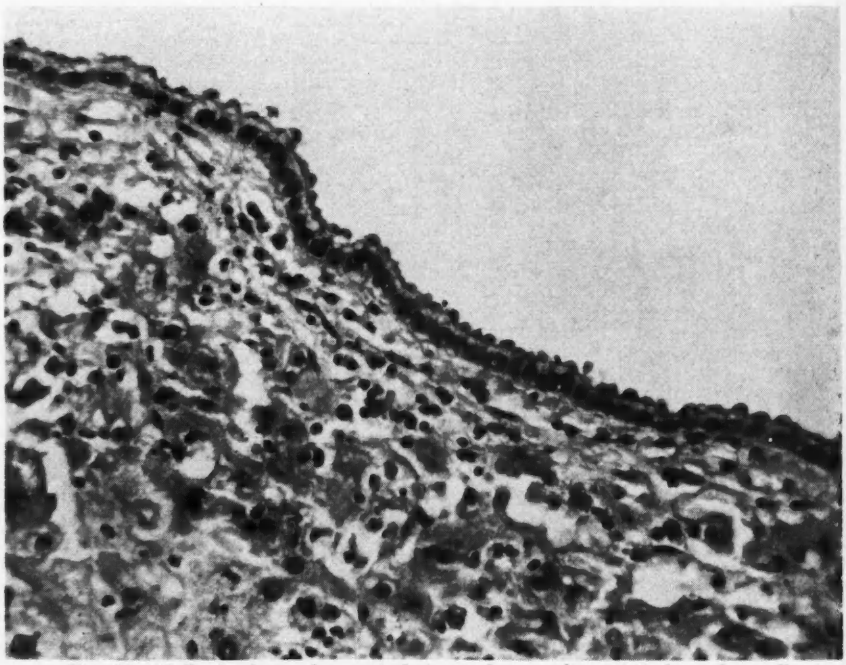


Fig. 25.—Wall of subcapsular cystic lesion in hamster liver: 340 days after pellet implanted (Group C; H & E, original magnification x 225).

and hematogenous metastasis and transplantation to the cheek pouches of normal hamsters.

Several concomitant hepatic alterations have been described including zonal and focal necrosis, portal oval cell proliferation, bile duct proliferation with fibrosis, and cystic lesions. Ligation of the cystic duct at the time of pellet implantation has been observed to have no demonstrable effect on the occurrence of these hepatic lesions.

ACKNOWLEDGMENTS

Dr. J. Gort, Dr. W. Pawluk, Dr. O. Retzer and Dr. S. Husain, Surgical Research Fellows, assisted in the surgical part of this project.

Mr. Eric Beamont and Mr. Ted Burton prepared the photographs.

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RÉSUMÉ

On décrit ici une méthode expérimentale permettant d'induire des cancers de la vésicule biliaire chez le "hamster" doré.

Les animaux utilisés étaient âgés de trois mois en moyenne, du sexe mâle. Ils furent divisés en trois groupes: groupe A chez qui on implanta des petites tablettes de methyl-cholanthrène dans la vésicule; groupe B où, en plus de cette implantation, on pratiqua une ligature du canal cystique; groupe C chez qui l'on ajouta aux deux interventions signalées dans le groupe B, une dissection et une séparation de la vésicule de son lit hépatique.

Sur 59 de ces animaux, 54 moururent spontanément, les cinq autres furent sacrifiés. L'autopsie fut pratiquée dans tous les cas: des prélèvements pour histologie furent faits avec fixation en formaline et inclusion à la paraffine.

Dans ces conditions, 70% des cas développèrent un processus tumoral dans les 400 jours qui suivirent l'implantation de methyl-cholanthrène; il ne semble pas y avoir de différence dans les incidences de chacun des trois groupes.

Microscopiquement, la grande majorité de ces tumeurs étaient des adénocarcinomes bien différenciés. Des formations papillaires furent souvent rencontrées, associées à des images kystiques. L'invasion du parenchyme hépatique et des tissus adjacents était un facteur fréquent. Des réactions inflammatoires, consistant en l'envahissement de la paroi vésiculaire par des lymphocytes et des cellules plasmatiques apparurent souvent, sans relation avec la formation de la tumeur.

La malignité des tumeurs ainsi obtenues fut démontrée par les métastases sanguines ou lymphatiques, et par la possibilité de transplanter ces cellules dans la poche jugale de "hamsters" normaux.

URETERO-ILEO-SIGMOIDOSTOMY: SOME OBSERVATIONS ON ITS LIMITATIONS AND DANGERS IN URINARY DIVERSION BASED ON EXPERIMENTAL STUDIES ON MONGREL DOGS*

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RADICAL pelvic surgery is commonly associated with loss or removal of the terminal portions of both ureters and in many cases total extirpation of the bladder. This has intensified the search for a practical and lasting method of urinary diversion to replace these damaged or extirpated structures. Hinman and Weyrauch[1] published an extensive review in 1937 of methods of transplantation of the ureters, dating back in origin as far as 1879. Brunschwig and Greenman[2] in 1959, stated "almost every conceivable method of urinary diversion has been tried experimentally and/or clinically in the past". Thompson[3] has aptly considered the implications of the search for an adequate method of urinary diversion in three phases – surgical, renal and electrolyte. It is the latter two phases that are now being investigated extensively.

The work of Swenson, Fisher and Cendron,[4] and Swenson and Fisher[5] on total replacement of aganglionic ureters in children by a loop of ileum, led us to the investigation of this problem described in this report. Would total replacement of the ureter be a safer procedure than partial replacement? The pelvis and the ileum would be of much more equal size. Stricture would be less common. The absorptive area of the ileum would increase the chance of electrolyte imbalance. We have proved experimentally that a loop of ileum, six inches to 12 inches long can be used quite safely to replace the ureter[6] and have demonstrated both experimentally (Figs. 1 and 2) and clinically, that a solitary kidney, with an ileal ureter, if not obstructed or infected, will sustain life in an individual or animal. A patient with a solitary hydronephrotic kidney of severe degree has survived three years with an ileal ureter attached to her lower dilated calyx (Fig. 3). Infection and electrolyte imbalance has been no problem.[6, 7]

Stimulated by these results, it was considered worthwhile to study similar procedures with a Bricker Pouch[8-11] attached to the sigmoid. Would a loop of ileum placed between the ureter and the sigmoid protect the kidney against ascending infection? Would electrolyte balance be maintained in cases in which no obstruction existed?

Originally, it was intended to transplant both ureters into a loop of ileum eight inches to 10 inches long, the proximal end being closed and the distal end attached by an end-to-side anastomosis to the lower sigmoid. At this time Turnbull and Higgins[12] reported a similar technique with the addition of a valve at the distal end of the ileum which was inserted into the sigmoid as depicted in their original drawing (Fig. 4). Using the technique of Turnbull and Higgins, five separate experiments were carried out to test the value of this procedure, the type of experiment changing as the investigation proceeded.

Anesthetic and intravenous therapy.– Intravenous Combutal® or Nembutal® were used in all of these experiments. During the operation a slow drip of 5% dextrose in water was administered.

Preoperative preparation.—Fluids only were administered for 48 hours before operation. A laxative was given the night before operation and a cleansing enema was given several hours preoperatively. In the first series of animals, no drugs were administered to sterilize the intestinal tract. In the last four experiments, one or two tablets of Polyneosal* were administered

*From the Department of Surgery, University of Manitoba; the Department of Surgery, St. Boniface Hospital, and the Abbott Clinic, Winnipeg, Manitoba.

*Polyneosal (Neomycin sulphate 70,000 mcg., Polymixin B sulphate 10,000 units; sulfamerazine 100 mg., sulfathiazole 100 mg., sulfanilamide 100 mg.).



Fig. 1.—The ureter has been completely replaced with a loop of ileum. The ileal ureter and bladder are distended with water to show the outline better.

every eight hours, according to the weight of the dog, for three days preoperatively and continued for at least 10 days postoperatively.

Bacteriological and biochemical studies.—In the last three experiments cultures were taken from the ileal bladder and sigmoid each time the bowel was opened. Electrolyte studies were carried out in the last four series.



Fig. 2.—The kidney has been bisected. The ileum is opened and a probe has been passed into the pelvis of the kidney.



Fig. 3.—A loop of ileum has been anastomosed to the lower calyx of the kidney proximally. Its distal end is attached to the bladder end-to-side. The ileal loop was passed through the mesentery of the ascending colon and anastomosed in an extraperitoneal position.

Experiment 1

Eight mongrel dogs ranging from 14 lb. to 30 lb. were used. An ileal loop eight inches to 12 inches long was isolated. The continuity of the ileum was established by end-to-end anastomosis. The proximal end of the ileal loop was closed. A valve was formed on the distal end of the isolated loop and this was inserted into the sigmoid just above the peritoneal reflection with 0000 chromic catgut and silk. The ureters were then divided and transplanted into the isolated ileal loop (Fig. 4). The abdomen was closed with chromic 0 catgut and the skin was closed with dermal sutures.

Three animals died before recovering from the anesthetic. These were considered anesthetic deaths. The remaining five died within four days. At postmortem examination no obvious cause for death was evident. Histological examination of the kidneys showed only a membranous glomerulitis, tubular degeneration and protein exudate into glomeruli and proximal tubules (Figs. 5 and 6).

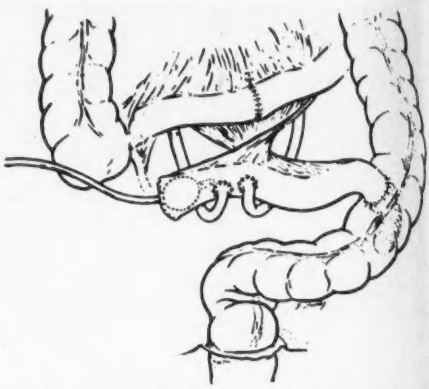


Fig. 4.—A diagrammatic sketch of Turnbull and Higgins' procedure (reproduced by permission of the authors and the publishers of the Cleveland Clinic Quarterly).

It was decided to change the approach to the problem by dividing the operation into stages. In addition, preoperative preparation of the bowel with Polyneosal was instituted. Following operation an antibiotic was administered intramuscularly daily until the animal tolerated oral therapy. The electrolytes were checked preoperatively and at staged intervals until the experiment ended.

Experiment 2

In a previous publication it was reported that an animal with one ureter completely replaced by an ileal loop anastomosed to the bladder, survived and showed no electrolyte imbalance after the opposite, normal kidney was removed.[6]

In the second experiment the following procedure was carried out in stages on two dogs. The ileal loop was isolated and inserted into the sigmoid as in the first experiment. At the end of one month or more, it was found that there was no evidence of edema in the ileal loop or valve. The loop was completely empty indicating good drainage. The right ureter was transplanted into the ileal loop. At the end of one month the electrolytes were found to

be normal. An intravenous pyelogram revealed a normal kidney on both sides. A barium enema revealed that there was no reflux into the ileal loop. The left kidney was then removed in both dogs.

In the first dog, No. 10, the blood urea nitrogen rose to 244 mg. % in seven days and to 270 mg. % on the 10th day. Muscular twitching, nausea, vomiting and loss of weight were marked. The animal died on the 10th day in obvious uremia. The second animal, dog No. 11, followed an identical course, the B.U.N. rising to 236 mg. % on the ninth day. This animal died on the 10th day.

An immediate postmortem examination was carried out on both animals. There was no infection in the peritoneal cavity. The ileal loop was intact and not distended. The kidney, ileal bladder and a portion of the sigmoid were removed and photographed. The postmortem findings in dog No. 11 are shown in Figs. 7 and 8. Fig. 9 is a photograph of an ileal valve protruding into the sigmoid. The pathological report on dog No. 11 was as follows: "The specimen consists of a kidney with attached ureter which is anastomosed to an ileal bladder. The distal portion of the ileal bladder is anastomosed to a segment of the sigmoid. The kidney is depleted of its capsule and measures 7.0 x 7.0 x 3.0 cm. The cortical surface is smooth and deep reddish brown throughout. On cut section, the cortex averages 6.0 mm. and the pyramids 1.5 mm. Both are moderately hyperemic but well demarcated. The renal pelvis is not distended and averages 3.0 mm. in diameter. The length of ureter present measures 13.0 cm. The uretero-ileal anastomosis is intact and patent from within the ileum. There is a small mucosal-like protrusion at the site of the anastomosis which is bluish-red in colour. The ureteral mucosa is smooth and glistening. The segment of ileum measures 12.0 cm. in length by 3.5 cm. in circumference throughout. There is slight loss of rugal folds throughout the ileum but no ulcerations are observed. The wall measures 0.4 cm. in thickness. The ileosigmoid anastomosis is patent. The mucosal junctions protrude within the

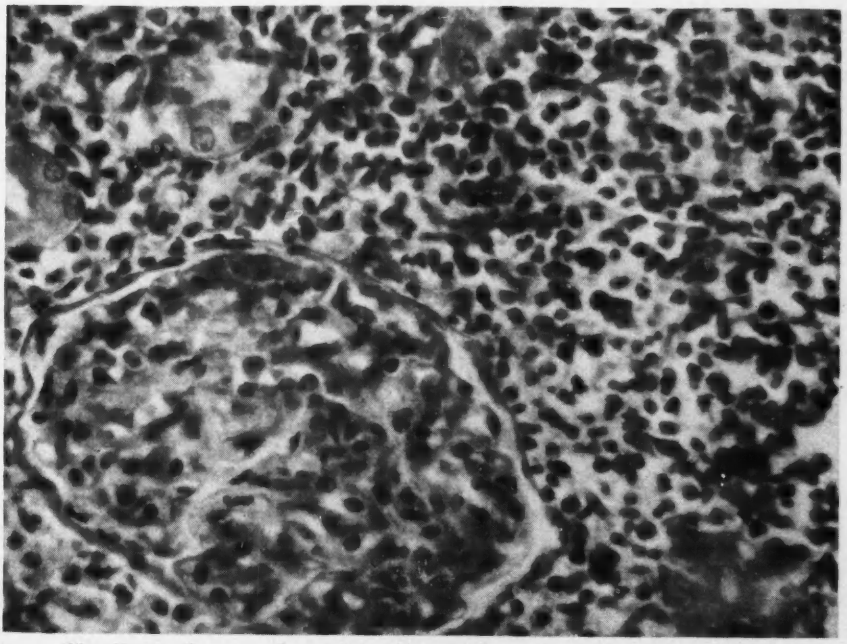


Fig. 5.—Membranous glomerulitis. The capillaries have thickened walls. The surrounding stroma contains chronic inflammatory infiltrate (original magnification X 400).

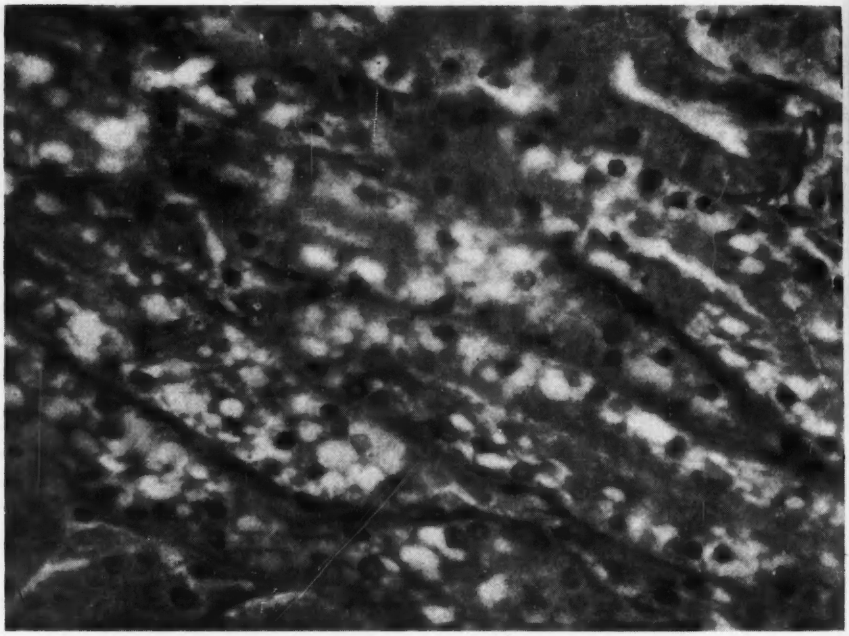


Fig. 6.—Renal tubular changes. There is vacuolation of the tubular epithelium and protein material within the tubular lumina (original magnification X 400).

sigmoid as a small nodule measuring 8.0 mm. in height. The sigmoid mucosa is slightly increased in redness but otherwise not remarkable along the margins of the anastomosis. The segment of sigmoid is 12.0 cm. in length by 5.0 cm. in circumference. There is slight flattening of the transverse mucosal folds but no ulcerations are noted. Attached to the ileum is a small amount of mesentery which contains engorged vessels."

Microscopically, the kidney sections showed protein transudate in Bowman's space and in the proximal convoluted tubules and there was mucosal atrophy of the ureter. The ileum and colon showed no microscopic abnormality.*

Bacteriology.—Cultures were taken under sterile precautions from the ileal bladder and kidney pelvis and a pure culture of *E. coli* was grown from both organs. It was sensitive only to Furadantin.®

*The autopsy report was prepared by Dr. M. Hamonic.

EXPERIMENT 3

In this series, preoperative and postoperative care, electrolyte and bacteriological studies were carried out as in Experiment 2.

In two animals an ileal loop was isolated. The continuity of the intestinal tract was re-established and the proximal end of the isolated loop was closed. A valve was formed at the distal end and inserted into the sigmoid. After a month or more the right ureter was transplanted into the ileal loop. The second ureter was transplanted in like manner after a similar period of time.

The first animal in this experiment (dog No. 12) lived seven days following transplantation of the second ureter. The B.U.N. rose from 20 mg. % to 169 mg. % before death. The electrolytes were otherwise normal.

The second dog (No. 14) lived for 28 days. The B.U.N. rose from 20 mg. % at the time of the second transplantation to 330 mg. % the day before death. Serum



Fig. 7.—Postmortem specimen of an ureteroileo-sigmoidostomy from an experimental dog (No. 11). Note the normal appearance of the ureter and kidney.

sodium rose from 119 mEq./l to 139 mEq./l. and the serum chlorides fell to 83 mEq./l. The CO_2 combining power fell to 6 mEq./l.

Postmortem examinations were performed on these animals directly after death.

Pathological report (dog No. 12).—"The specimen consists of colon with laterally anastomosed ileal loop. Both right and left ureters are included and are anastomosed laterally to the ileal loop and the attached kidneys are also present. The left kidney is small (approximately one-half the size of the right) and measures 3.0 cm. in length by 1.0 cm. in width by 1.2 cm. in thickness. Its capsule is thin and strips readily. The cortical surface is small, deep reddish-grey and on section the cortex averages 0.6 cm. in thickness. It is well demarcated from the adjacent medulla which is pale grey. The pelvis is not dilated. The ureter is patent except at a point 3.0 cm. from its site of anastomosis, where it is thickened and narrowed to less than 0.1 cm. The distal portion beyond the narrowing is collapsed and the anastomotic site cannot be identified. The right kidney measures 5.0 x 3.0 x 2.0 cm. and does not appear remarkable. The right ureter is patent and the anastomotic opening is not remarkable. The ileal loop contains mucoid-like material and the mucosa is somewhat flattened. The ileal-sigmoid anastomosis is patent. The sigmoid segment is not remarkable."

Protein tubular casts were noted in sections of the right kidney. The ileum and colon showed no microscopic abnormality. The pathologist's diagnosis was "Chronic peri-ureteritis with luminal obstruction, left."

Photographs were taken of the specimens in this case and are shown in Fig. 10.

Pathological report (dog No. 14).—"Sub-

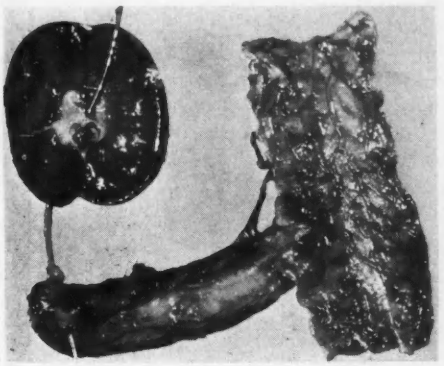


Fig. 8.—The kidney has been bisected and appears grossly normal. The ileal bladder and sigmoid have been laid open. A probe has been passed from the kidney pelvis, down the ureter into the ileal bladder.



Fig. 9.—Actual photograph of an ileal valve extending into the sigmoid, using the technique of Turnbull and Higgins.

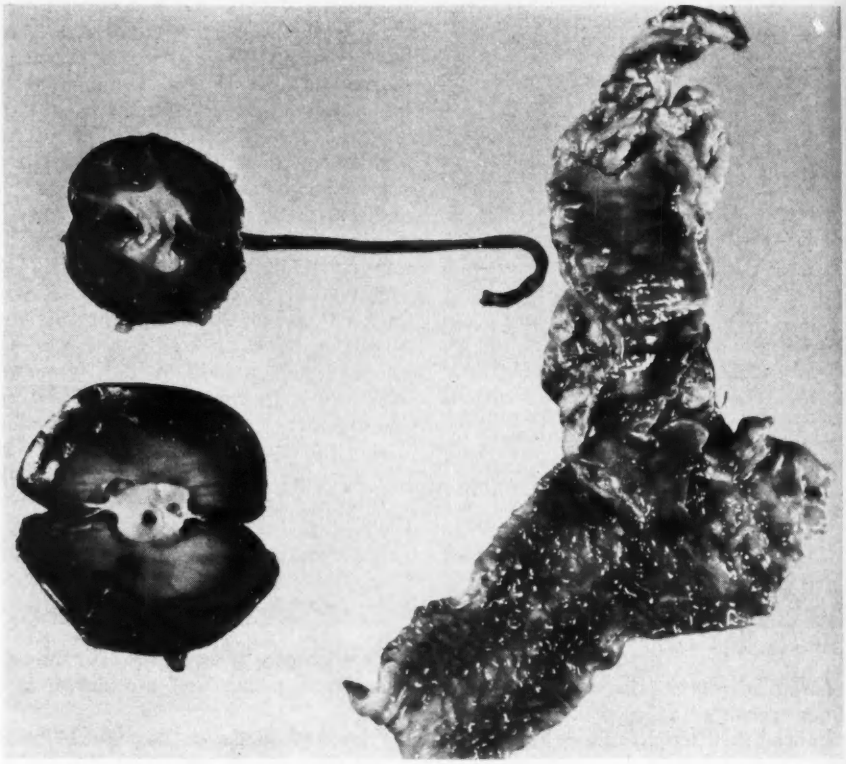


Fig. 10.—The stricture at the lower end of the ureter was considered to be a surgical error at the time of transplantation.

mitted are two kidneys with attached ureters anastomosed to an ileal loop bladder. The latter is anastomosed to the sigmoid colon by an end-to-side anastomosis. The right kidney shows no evidence of hydronephrosis but scattered throughout are zones of hemorrhage and regular small foci which are pale and probably correspond to abscesses. The ureter is patent. The ileal loop and sigmoid are not remarkable. The left kidney shows moderate hydronephrosis and pus within its pelvis.

Diagnosis.—Acute bilateral necrotizing papillitis, pyelonephritis and ureteritis (Figs. 11, 12 and 13). Unilateral moderate hydronephrosis due to external obstruction of the ureter by a fibrous band.

Bacteriology.—Culture from the ileal bladder at the time the second ureter was transplanted showed a few saprophytic staphylococci and *E.coli.* At the time of death only *B.proteus* was cultured.

EXPERIMENT 4

Eight animals were prepared as previously described. An ileal bladder with a Turnbull valve was formed at the first stage and inserted into the sigmoid colon. Bilateral ureteral transplantation of both ureters was performed after one month or more. It has been previously shown that all edema disappears from the loop and valve during this period and that there is good drainage established from the loop into the sigmoid. The possibility of the ureters draining into a closed loop has been avoided.

Electrolyte studies were conducted preoperatively and at staged intervals until the animals succumbed. Cultures from the ileal bladder were taken at the time of ureteral transplantation and also from the kidneys and ileal loop at the time of death.

In seven animals the postoperative course was similar. The B.U.N. rose gradually but

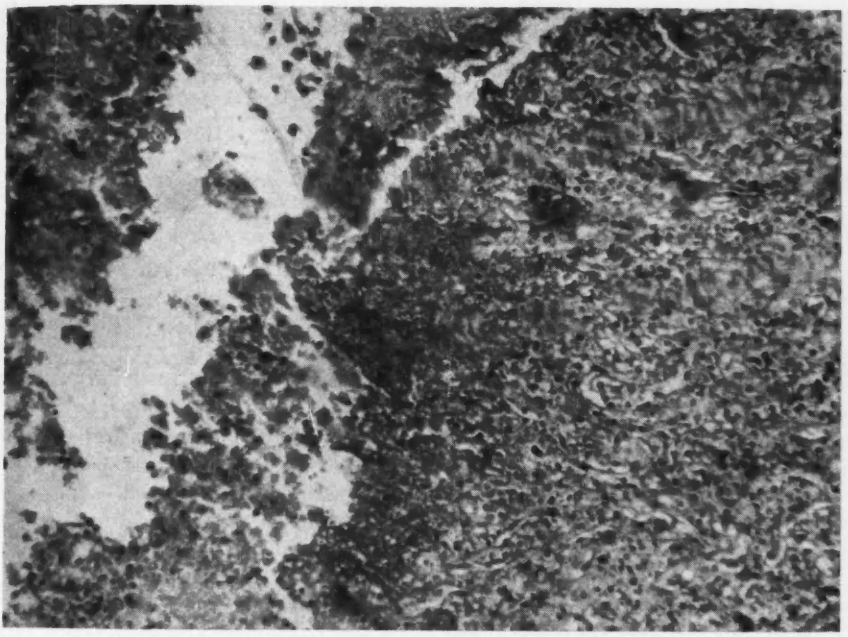


Fig. 11.—Necrotizing papillitis. Note ulceration of the renal papillae and the acute inflammatory reaction present (original magnification X 150).

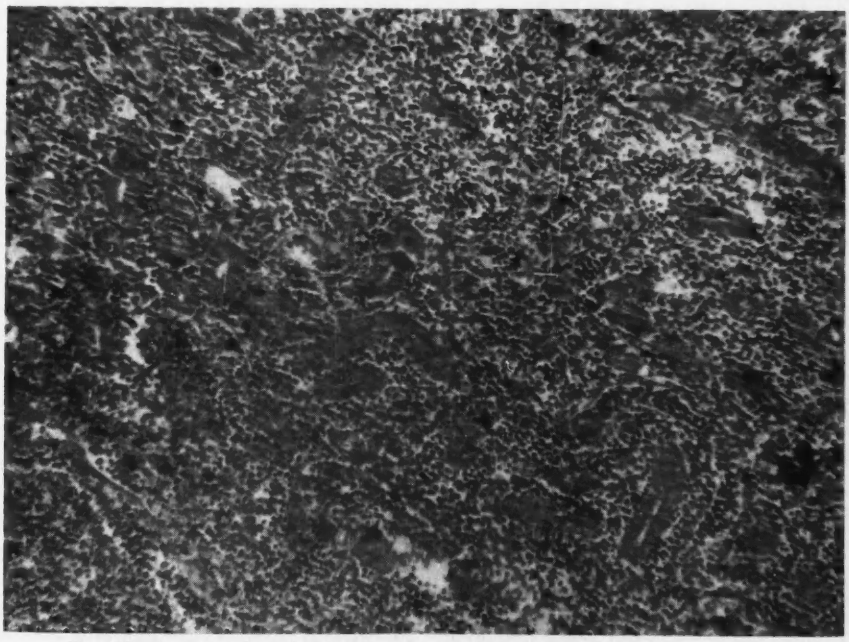


Fig. 12.—Acute pyelonephritis. The interstitial tissues are diffusely infiltrated by acute inflammatory cells (original magnification X 150).

TABLE I.—Progressive Electrolyte Changes Following Total Replacement of Bladder by Ileal Loop (Uretero-Ileo-Sigmoidostomy): Bilateral Transplantation of Ureters (Second Stage)

Number	Blood urea nitrogen mg.%		K mEq./l.	Na mEq./l.	Cl mEq./l.	Survival (days)	CO_2 mEq./l.
13	4		3.9	135	110		20
	16		4.8	143	102		18
	93		3.9	136	102		13
	70		3.5	138	110	26	
	D						
15	17		5.1	148	102		23
	14	60	4.5	144	107		14
	22	46	3.9	139	113		13
	25	—	2.9	178	110	31	
		D					
16	20		4.6	143	105		22
	22		4.8	144	106		24
	74		4.6	140	110		18
	102		4.8	138	112	30	16
	D						
20	11		4.5	145	106		24
	20		4.7	142	99		28
	20		3.5	139	104		14
	50	42	4.3	158	136	2½	10
	52	—		148		months †	
		D					
23	16		5.0	144	103		28
	16		4.8	147	113		25
	84		4.9	140	115	10	25
	D						
25	14	156	4.4	146	105		23
	10	84	4.2	146	110		20
	134	—	3.8	137	105	22	14
		D					
26	17		3.0	140	108		19
	10	170	3.6	142	110		20
	84	146	3.6	146	120		15
	142	—	3.2			26	16
		D					
32	16		4.0	134	112		20
	20		4.3	140	102		18
	68		3.8	135	100		15
	82		3.2	129	105	16	11
	D						

The first figure in each column represents the electrolyte estimation preoperatively.

The second figure in each column represents the electrolyte estimation at the time of ureteral transplantation.

The succeeding figures represent periodic electrolyte studies during the interval before death.

Key—Survival—Duration of survival following ureteral transplant.

†—Dog No. 20 lived two and one-half months and died suddenly.

D—Died.

not so fast or as high as in the animals in which ureteral transplantation was performed at staged intervals (Table I). Serum chloride and sodium levels remained within normal limits. The potassium fell to a low normal and the CO_2 combining power fell in all cases, the average being 13.8 mEq./l. The average duration of life in the seven animals following ureteral transplantation was 23 days.

The eighth dog (No. 20) followed a different course. There was an interval of seven weeks between the formation of the ileal bladder and the ureteral transplantation. The B.U.N. at the first stage was 11 mg. % and at the second stage 20 mg. %. This was checked for the next six weeks at weekly intervals and was as follows: 26, 50, 52, 42, 36 and 32 mg. %. The CO_2 combining power fell as the B.U.N. rose and rose as the B.U.N. fell. The animal was gaining weight, looked better and ate better. Electrolyte studies were then temporarily discontinued. The animal died suddenly two and one-half months following ureteral transplantation.

Postmortem findings.—The right kidney was a huge thin-walled hydronephrotic sac. The ureter was dilated. The left kidney appeared grossly normal. The entire system was removed for photographing and pathological study. Cultures were taken. Figs. 14 and 15 illustrate the pathological findings.

Pathological report (dog No. 20).—"*Gross.* The specimen consists of a right kidney which is markedly hydronephrotic and composed essentially of a thin, flattened cortex 4.0 mm. in thickness. The kidney measures 4.5 x 3.0 x 2.0 cm. The pelvis is 3.0 cm. in length and 2.0 cm. in width. There is distention of the right ureter which appears to be totally occluded at its site of anastomosis with the ileum. However, under forceful pressure on the ureter, water can be passed through the latter opening. The left kidney measures 5.0 x 3.0 x 2.0 cm. and is not hydronephrotic. However, the left ureter is slightly distended and its os is slightly narrowed as well. The narrowing at the uretero-ileal anastomosis is due to thickening of the walls of the ureter. Also submitted is a segment of ileum. A separate segment of colon is also present".

Microscopically the right kidney showed marked hydronephrosis accompanied by



Fig. 13.—Acute ureteritis. The mucosa is ulcerated and an acute and chronic infiltrate is seen throughout the wall associated with edema and early fibrosis (original magnification X 150).

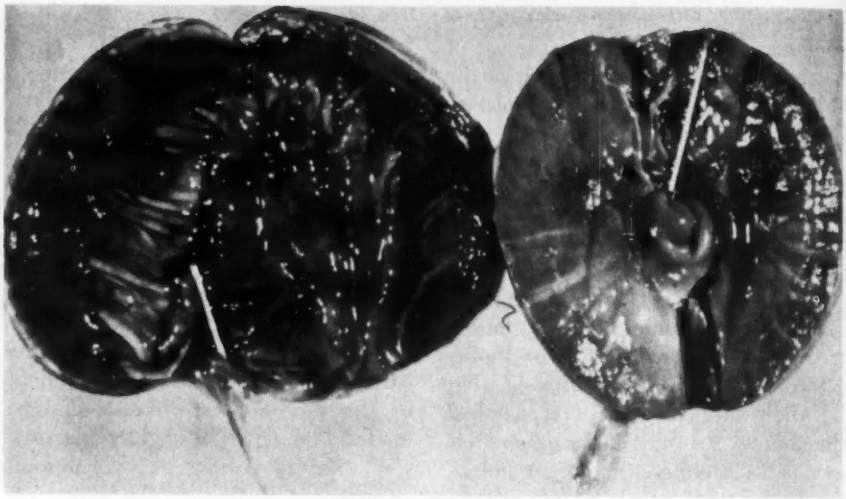


Fig. 14.—The right kidney is a huge hydronephrotic sac due to a stricture in the lower end of the ureter.

hydroureter, acute pyelitis and ureteritis. There was superficial atrophy of the ileal mucosa. The left kidney showed slight hydroureter, chronic pyelitis and ureteritis.

EXPERIMENT 5

On November 17, 1959, an ileal loop was formed and attached to the sigmoid in dog No. 21 which weighed 22 lb. Bilateral

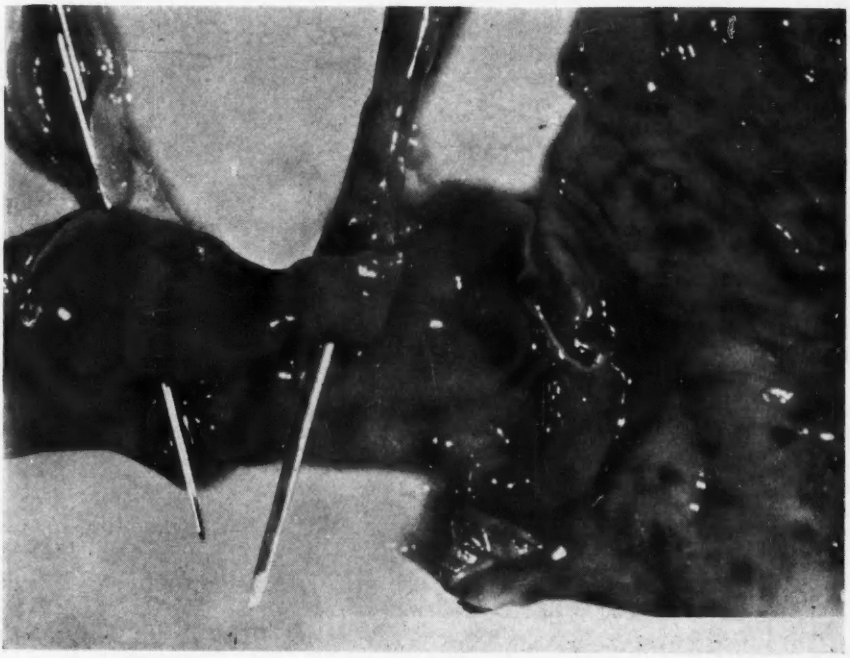


Fig. 15.—The ureter on the right is grossly dilated. There is a firm hard stricture just proximal to its insertion into the ileal bladder. It was not complete as a fine probe passed through it.

ureteral transplantation of both ureters was done on December 22. On December 28, the B.U.N. was 50 mg. %. Repeat serum electrolyte studies on January 18 gave the following results: B.U.N. 76 mg. %, potassium 3.0 mEq./l., sodium 145 mEq./l., chlorides 120 mEq./l., CO_2 combining power 13 mEq./l. The animal's weight had fallen to 18 lb. and it was in poor condition. On January 19 the ileal loop was disconnected from the sigmoid, and an end-to-side anastomosis of the ileal loop to the bladder was performed. On March 30, the animal had regained its normal weight, the B.U.N. had fallen to 28 mg. % and the dog looked well. On June 25, the B.U.N. was 22 mg. % and the animal weighed 23 lb.

Discussion

The rapid occurrence of death after operation in the first series of dogs is noteworthy. In the Bricker procedure open drainage of the loop is established at the original operation. In Turnbull and Higgins' operation, the loop is decompressed by inserting a Foley catheter into the proximal end of the isolated ileal loop. This is not possible in animal experiments unless the animal has been trained to survive in a sling. The institution of a two-stage operation appeared to have the same effect at least to a considerable degree. We felt that in the first series of dogs, edema and constriction at the insertion into the sigmoid, together with the constrictive effect of a valve at the distal end of the ileum, converted the ileal bladder into a closed loop. The deaths were undoubtedly due to biochemical disturbances rather than to infection.

Barium enemas were done in four dogs to test the efficacy of the ileal valve in preventing reflux into the ileal bladder. There was no reflux in any of the four animals and thereafter the procedure was discontinued.

In four dogs not included in this series, the ileal loop came out of the sigmoid and general peritonitis followed. This was considered to be a technical error.

In three dogs the ileal bladder underwent intussusception into the sigmoid and protruded from the rectum. Two of these animals were sacrificed. Dog No. 20, one of the animals with this complication, was subjected to re-operation. The ileal bladder and part of the sigmoid were resected and the sigmoid was then re-anastomosed by an end-to-end anastomosis. The abdomen was closed. The animal did very well and was used six weeks later for the same experiment. As a result of this complication the ileum was thereafter stitched to the side of the sigmoid for one inch proximal to its insertion. This appeared to prevent further instances of intussusception of the ileal bladder into the sigmoid.

In one dog a huge pyosalpinx associated with mild peritonitis was found. This animal was omitted from the series.

In the last four series of experiments, all animals developed an ascending infection, including the animal that survived after disconnecting the ileal bladder from the sigmoid and reimplanting it into the bladder proper. These infections varied from a chronic pyelitis to an acute ascending ureteritis, pyelitis and pyelonephritis. It would therefore appear that the valve does not prevent the proximal extension of infection from the sigmoid.

Summary

This is a preliminary report. It is realized that the number of experimented subjects in this series is not sufficiently large to justify definite conclusions but it is considered that the following pertinent observations are in order.

Obstruction, infection, and reabsorption are the principal causes of renal failure after creation of an ileal bladder.

Either a two-stage procedure or decompression of the ileal loop will prevent obstruction from edema at the site of insertion into the sigmoid. Careful anastomosis may prevent ureteric strictures such as those that caused failure in the most promising case (dog No. 20) in the study reported here.

In dogs, the peristaltic action of the ileum does not prevent ascending infection (but this problem mainly causes serious trouble where there is stricture formation and is not inescapable).

In the absence of mechanical or infectious complications, uremia seems to be due to reabsorption of urea and hydrogen, the absorptive surface after uretero-ileo-sigmoid anastomosis being much greater than that created by uretero-ileal diversion of urine.

The changes due to reabsorption are reversible. Disconnecting the ileal loop from the sigmoid and reimplanting it into the bladder restores the animal to health and electrolyte balance. One animal was able to restore efficient excretion of urea and acid with the ileal bladder still anastomosed to the sigmoid.

Mucus plugs have caused no trouble in these experiments with ileal loops.

ACKNOWLEDGMENT

This research problem was carried out on Grant No. S257 from the Banting Research Foundation.

The bacteriological, pathological, radiological, photographic, biochemical work and anesthetics were generously provided by the various departments of St. Boniface Hospital.

The firm of Johnson & Johnson supplied a great proportion of the suture material. Diagram (Fig. 3) was drawn by Miss Joy, medical artist.

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RÉSUMÉ

La chirurgie radicale du pelvis se voit souvent entraînée dans des résections partielles des bouts terminaux des uretères des deux côtés et aussi de la vessie. Ceci a naturellement conduit à intensifier les recherches en vue de trouver un moyen pratique de détournement des urines. Différents moyens on été proposés pour ce but. Les auteurs ont procédé à des séries d'expériences sur des chiens à la même fin.

Dans une *première série*, on isola une anse d'iléon de huit à 12 pouces de long; la continuité de l'intestin fut rétablie par une suture termino-terminale; le bout proximal de l'anse iléale fut fermé; sur l'extrémité distale on forma une valvule et l'on inséra le tout dans le sigmoïde juste au-dessus de la ligne de réflexion du péritoine. Les uretères furent alors sectionnés et transplantés dans cette boucle. Tous les animaux d'expérience moururent dans les quatre jours subséquents. Il fut alors décidé de procéder à ces essais par paliers.

Deuxième série.—On procéda dans un premier temps à l'isolation et à l'insertion de l'anse iléale. Un mois plus tard, on fit l'implantation uretérale à droite. Un autre mois plus tard le pyélogramme intra-veineux et le lavement baryté étaient normaux. On enleva le rein gauche. Les chiens moururent rapidement d'urémie.

Troisième série.—On procéda comme dans la série 2 pour le premier temps. Un mois plus tard on inséra l'uretère droit dans l'anse iléale et un autre mois plus tard on fit de même pour l'uretère gauche. Les chiens moururent de pyélonéphrite et d'uréterite bilatérales.

Quatrième série.—On procéda ici comme précédemment, mais les implantations uretérales furent faites simultanément et plus tardivement. Ici, les suites furent variables selon les animaux.

Cinquième série.—Les premiers temps furent ici conduits comme dans les séries précédentes. L'évolution post-opératoire ressembla en tous points à ce qui a été précédemment décrit. Cependant, après trois mois, l'animal ayant un état général médiocre, on décida de supprimer l'anastomose iléo-sigmoïdienne et de réimplanter l'anse iléale dans la vessie. L'état de l'animal s'améliora rapidement.

Il ressort de l'ensemble de cette expérimentation que, en dehors de toute complication infectieuse et de toute obstruction mécanique, l'urémie apparaît; elle est causée par la réabsorbtion d'urée au niveau de la muqueuse iléale. Ceci semble être bien démontré par la cinquième série expérimentale: les phénomènes apparaissent comme réversibles. En effet, après installation d'un état urémique, celui-ci peut être supprimé par la réimplantation de l'anse iléale dans la vessie.

SURGICAL TECHNIQUE

INCISIONS, LACERATIONS AND SCARS

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THE AVOIDANCE of poor scars is both functionally and cosmetically important to any patient. These can be simply classified as follows:

TYPES OF POOR SCARS

a. Movement scars.
b. Tension scars.
c. Depressed and fixed scars.
d. True keloids.
e. Hypertrophic scars (pseudo-keloids).

The most important and most common of these is the hypertrophic scar; therefore, only brief reference will be made in this report to the other types of scarring.

1. *Movement scars.*—Movement is a well recognized factor in the production of thickened scars which do not occur as a result of true hypertrophy. They are raised above the surface, particularly when crossing a concavity. They are not abnormally vascular and do not tend to regress, but to persist as tight bands of adult fibrous tissue, the epithelial covering of which is frequently keratotic. Movement tends to produce cracking which is followed by ulceration. The increasing amount of scar tissue produced in healing creates a vicious circle.

2. *Tension scars.*—Unrelenting mild tension such as that placed on a scar at right angles to Langer's lines often results in a gradual widening without surface breakdown. The end result is a flat atrophic shiny scar which may be almost as wide as the original surgical defect.

3. *Depressed and fixed scars.*—Depressed and fixed scars are the result of almost purely physical factors which result in fixation to underlying rigid structures such as bone, muscle or the trachea. They are more directly related to the location and degree of trauma than to skin lines and they may be remedied by removal of the unyielding fibrous tissue and the interposition of soft padding such as fat between the skin and the underlying fixed structures.

4. *True keloids.*—True keloids occur frequently in Negroes but are rarely seen in other races. Up to the end of the first month, they are indistinguishable clinically and microscopically from hypertrophic scars. A history of keloid formation in former scars may be of value. The true keloid does not regress and it may continue to increase in size over an almost indefinite period and extend beyond the confines of the original scar (Fig. 1).

5. *Hypertrophic scars (pseudo-keloids).*—This type is the commonest type of poor scar, and may be seen after surgical incisions, accidental lacerations, deep second degree burns and indeed almost any form of skin damage. The scar passes through a series of recognizable phases and though these vary in duration and in degree, the pattern produced is quite constant (Fig. 2).

Clinical phases of hypertrophic scars

Primary healing occurring in three to five days results in a faintly pink line on a level with the surrounding skin. In the next 20 to 30 days, the scar increases in width and prominence, the pinkish colour becomes more marked and the surface becomes shiny and has a "moist" appearance. It is almost always described by the patient as "very itchy".

Up to this point a hypertrophic scar is identical histologically and clinically with a true keloid.

The hypertrophic scar undergoes a series of changes which may occur over a period of six to 60 months in untreated cases. During this time it loses its "moist" appearance, the reddish-pink colour fades, it decreases in bulk and prominence and finally becomes a thin, white, flat, slightly wrinkled scar.

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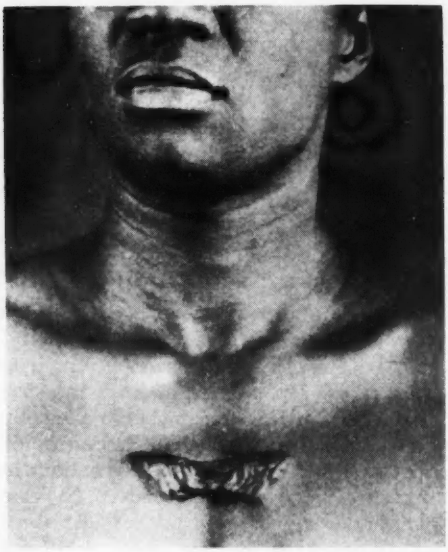


Fig. 1a

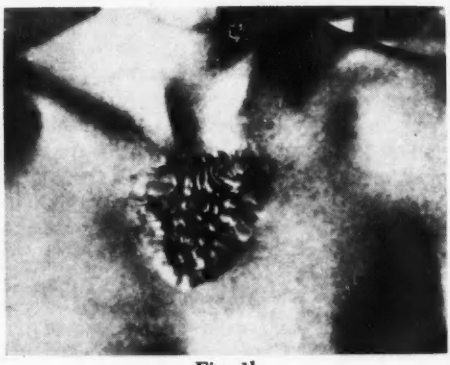


Fig. 1b

Figs. 1a and b.—True keloids occurring spontaneously in the classical pre-sternal location in young Negro woman. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

Factors that Influence the Type of Scar Formation

1. *Types of skin—"Keloid formers".*—Negroes and reddish haired people have long been recognized as individuals with a tendency to form poor scars. Those with thyroid dysfunction and endocrine disturbance are prone to exhibit similar changes. Children with a lot of lanugo hair ("baby hair"—"baby fuzz") are noteworthy in this regard. Lastly, a miscellaneous group of persons who do not fit into any of the foregoing categories are equally likely to present the same problem. These groups will be discussed later in this paper, in the light of recent knowledge.

2. *The problem of Langer's lines or wrinkle lines.*—In 1861, Langer,[1] an Austrian anatomist, published his monumental work on the subject of lines of skin tension (Figs. 3 and 4). Kocher in 1892, suggested that surgical incisions should follow Langer's lines and in 1907 published the description of his classical incisions drawn over these lines.[2] In 1935, Webster[3] recorded his disagreement with Kocher's advocacy of vertical incisions across joints, and midline incisions, and expressed the opinion that natural wrinkle lines are much more advantageous sites for skin incisions. In 1947, Shaw and Copenhaver[6] published an excellent review of the subject, and presented the results of their original research which clearly demonstrated that in most areas the majority of fibres of connective tissue follow patterns similar to those of Langer's lines. More recently Kraissl,[11] in an excellent article published in 1951, reviewed and challenged Langer's and Kocher's concepts and emphasized that wrinkle lines are frequently more important than the classical Langer's lines (Figs. 5, 6 and 7). Quoting Webster, Kraissl observed that "The simplest rule for making incisions in the most favourable direction is to follow the natural wrinkle lines. These are usually recognizable in the face, the neck, at the wrist, the axilla, the groin, or the back of the knee". Conway[4] in 1938 noted the differences in physiologic elasticity in the same areas of the body between live subjects and the cadavers on which all the early work was based.

Kraissl makes a fundamental observation when he says "It is evident that these (wrinkle) lines fall in a pattern across the muscles perpendicular to their action" (Figs. 7, 8, 9, 10 and 11).

The excellent diagram of elective surgical incisions of the hand published by Bruner[7] in the British Journal of Plastic Surgery and illustrated in Fig. 13 is worthy of note.

3. *Primary healing.*—It is universally agreed that primary healing of any wound produces a minimal amount of scarring.

4. *Infection.*—Gross infection, resulting in healing by secondary intention, which is always delayed healing, is followed by the laying down of excessive fibrous tissue.

5. *Tension beyond physiological limits.*—Closure of a wide wound under considerable tension often results in strangulation of blood supply by the sutures, necrosis of the wound margins and subsequent secondary healing.

6. *Motion.*—"Rest of the part" at the time of wound healing is of the utmost importance. Later, after healing, physiological motion, such as the tension on the skin of web spaces in abduction of fingers, can cause perpetuation of heavy scarring. If the skin tension is relieved by the addition of skin without removal of all of the old scar, the latter will usually regress without further therapy.

7. *Keratin sensitivity.*—The experimental animal studies of Glücksmann[9] at Cambridge, which were corroborated clinically by Mowlem[8] indicate that the presence of keratin in a wound may well be an important factor in the production of hypertrophic scar formation. These observations constitute the most significant and outstanding recent contribution to the literature on this subject. After implantation of small autogenous grafts beneath the skin of laboratory animals, these workers observed that "so long as the keratin produced by the graft and its contained hair follicles remained encysted no tissue response to the presence of the implant was discernible. As soon, however, as the cyst wall perforated exposing the subcutaneous tissue to the effect of the keratin, there was a dilatation of those vessels in the immediate neighbourhood, with perivascular infiltration of round cells. The process continued until the keratin had been removed by the normal scavenging mechanism."



Fig. 2.—A laceration developing to a hypertrophied scar in a young man with abundant facial growth of lanugo hair. (Courtesy of Mr. Rainsford Mowlem. Reproduced by kind permission of the British Journal of Plastic Surgery).

Mowlem has demonstrated clearly the close relationship between these histological changes and the commonly observed cycle of changes through which a hypertrophic scar passes.

Keratin Factors

(a) *Systemic.*—It is reasonable to assume that the majority of wounds at some stage contain some foreign material, yet all do not form hypertrophic scars. It is also reasonable to assume that the amount of keratin buried within the wound will vary, as will the individual systemic reaction to keratin. However, the systemic factor will be constant in any individual patient. Clinically, one observes that two scars in the same patient do not produce similar and equal responses.

(b) *Local.*—The local response is confined to the region of the blood vessels which are exposed to the effects of the keratin (Fig. 12).

Sources of Buried Keratin

1. *Lanugo hair.*—Lanugo hair is the fine abundant baby hair or "baby fuzz" which is often present in great quantity. Mowlen emphasizes its fineness, the great numbers of individual hairs, their slow growth and most important, the *obliquity* of their growth. Therefore, the number of buried hair follicles will depend on whether the incision is parallel to, or at right angles to, their general direction. Beard or scalp hair is much less dense and grows more vigorously and rapidly so that most hair follicles of this type will only be temporarily buried.

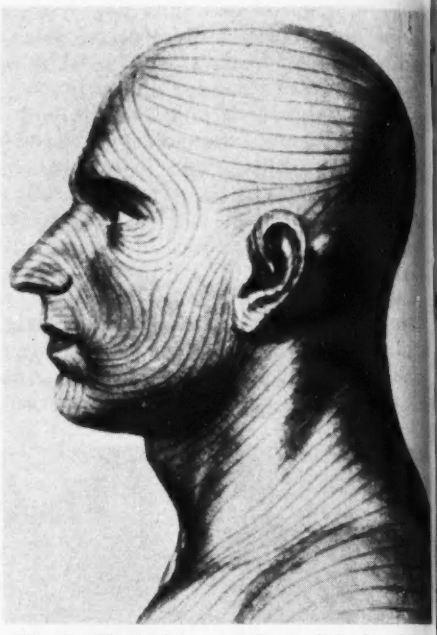


Fig. 4.—Classical Langer's lines of the head and neck. (Courtesy of Dr. C. J. Kraissl after Langer. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

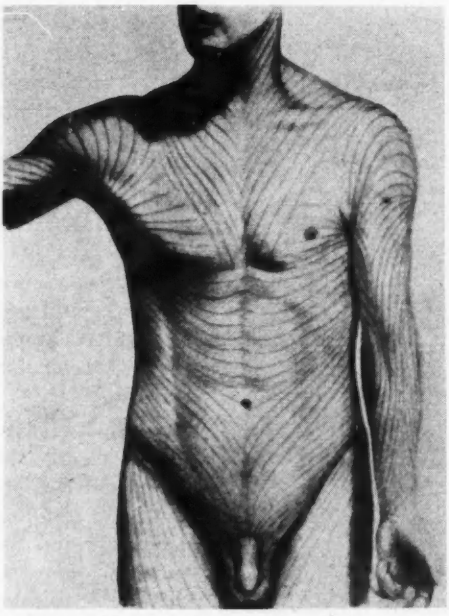


Fig. 3.—Classical Langer's lines of the anterior body. (Courtesy of Dr. C. J. Kraissl after Langer. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

2. *Sebaceous glands and sweat glands.*—Both sebaceous and sweat glands are rich sources of abundant keratin. It is noted clinically that areas that show profuse sweating, such as the palms of the hands, have a tendency to hypertrophic scarring.

3. *Skin thickness.*—It seems reasonable to assume that the chance of burying these skin appendages is proportionate to the thickness of the skin. This assumption is borne out clinically, as it is rare to see a hypertrophic scar in the skin of the eyelids, an area of the body in which the skin is thinnest.

Types of People—Bad Skin

It seems reasonable, therefore, to recategorize people with "bad skin" in the light of this new knowledge.



Fig. 5a

1. *Individuals with red hair and fair complexion.*—These persons usually have an overabundance of lanugo hair.

2. *Individuals with thyroid or endocrine disturbance.*—Such people have coarse skin with excessive terminal hair and an active sebaceous gland mechanism.

3. *Negroes.*—Characteristically, Negroes have an oily skin with many active sweat glands. Mowlem suggests that African tribal marital selection, based on the presence of keloid scars, could accentuate the systemic factors in these individuals.

4. *Children.*—Many children have an abundant amount of lanugo hair. This is particularly noteworthy in the case of prepubertal boys.

5. *Adults.*—There are certain adults who do not fit into any of the aforementioned groups. However, one can frequently find evidence to suggest that the presence of keratin from some source is a factor contributing to hypertrophic scar formation in such individuals.

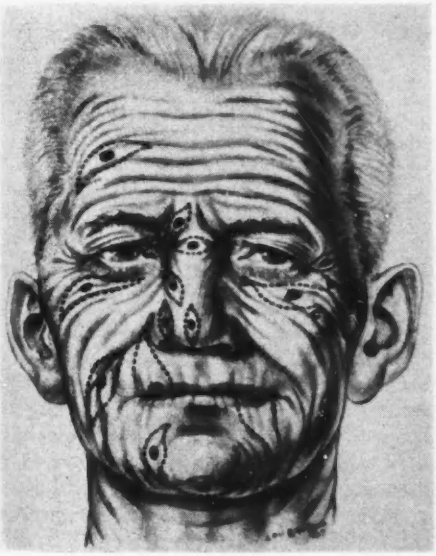


Fig. 5b

Figs. 5a and b.—A case photograph and its drawing with appropriate lines for excision of various lesions superimposed on the wrinkle pattern. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).



Fig. 6.—The same wrinkle pattern superimposed on a diagram of facial musculature demonstrating that wrinkle lines occur at right angles to the direction of pull of muscles. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).



Fig. 7.—Wrinkle lines of the side of the head and neck. Contrast this with Langer's lines (Fig. 4). (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

Control of Hypertrophic Scars

The following measures are suggested with the objective of reducing the possibility of hypertrophic scar formation.

1. In elective surgical procedures, select the proper wrinkle lines for incisions.

2. In the case of a traumatic wound that does not follow wrinkle lines, debride it carefully, convert it to proper wrinkle lines if possible, but above all strive for clean primary healing.

3. Do not use vertical incisions across flexion creases, but follow the wrinkle lines (flexion creases) and extend the incisions vertically where necessary, in areas where they are not subjected to extremes of tension and movement.

4. Avoid the inclusion in wounds of such foreign material as wisps of cotton from sponges, powder from gloves, or dirt from the road. It is of equal or even greater importance, in elective surgical procedures, to avoid the inclusion of keratin-forming skin appendages by skiving or irregular incisions, overlapping edges and so on.

5. In the case of children, avoid or delay non-essential operations where possible because of the tendency of their lanugo hair factor to produce heavy scars.

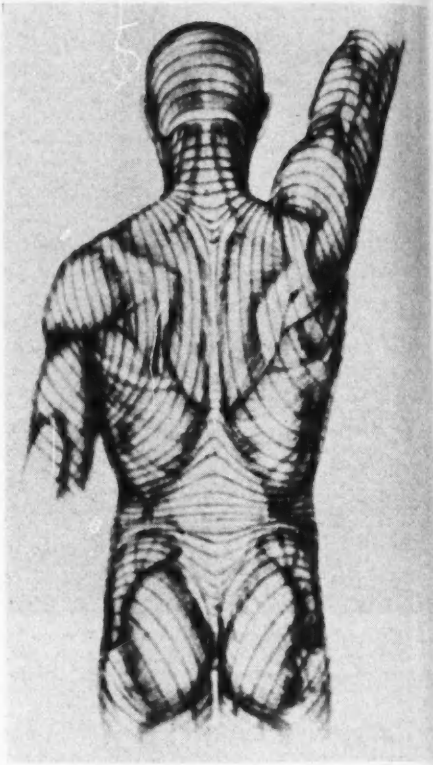


Fig. 8.—Wrinkle lines of posterior body. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

Treatment of Hypertrophic Scars

Once developed, hypertrophic scars may require treatment owing to associated itching or burning pain, interference with function or unsightly appearance. Even though circumstances surrounding the healing of the original wound did not favour clean primary healing, the latter may be achieved in some cases after simple surgical excision or revision to remove excessive fibrous tissue. However, under many circumstances a purely surgical approach will result in the ultimate formation of a scar of the same, or even larger size, because some of the aforementioned factors can not be adequately eliminated. In these situations radiotherapy may be of value. The objec-

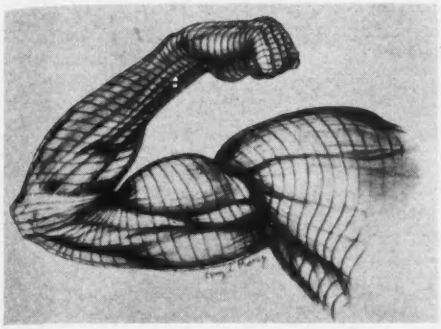


Fig. 9.—Wrinkle lines of the upper extremity when flexed. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

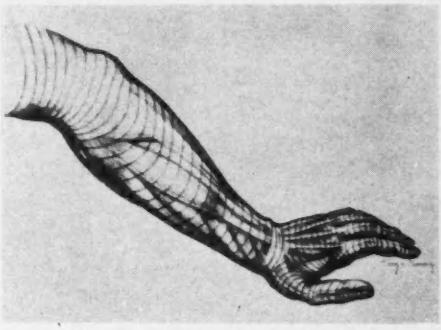


Fig. 10.—Wrinkle lines of the upper extremity when extended. (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

tive of such radiation is to make the keloid or hypertrophic scar smaller, softer, flatter and less obvious, more quickly than if resolution is allowed to occur without treatment. Very often the relief of itching and burning can be achieved quite promptly by this means. Because it induces softening, radiation may lessen the extent of minimal contractures but will not appreciably affect more extensive types. Where

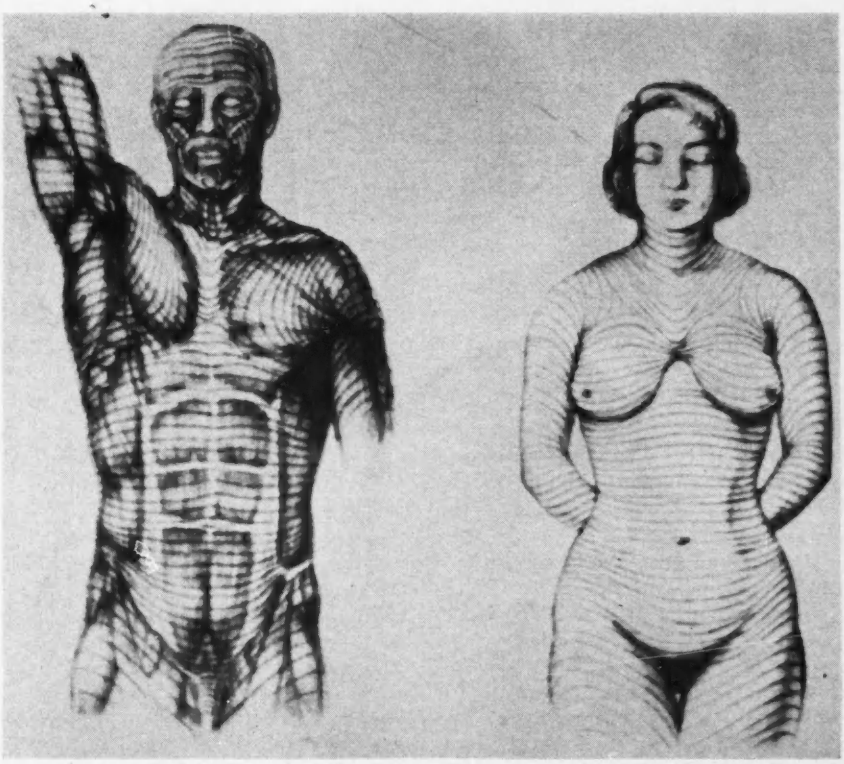


Fig. 11.—Wrinkle lines of the anterior body, male and female. Contrast this with Langer's lines (Fig. 3). (Courtesy of Dr. C. J. Kraissl. Reproduced by kind permission of the Journal of Plastic and Reconstructive Surgery).

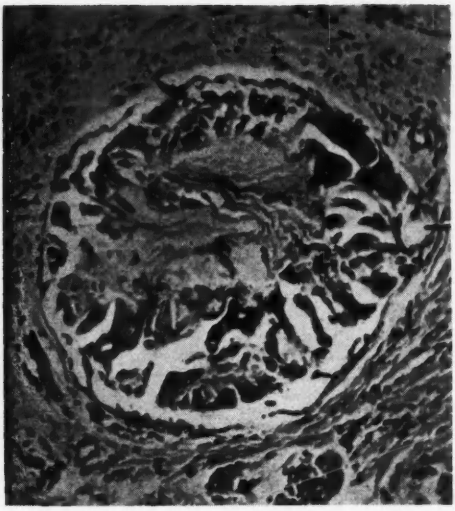


Fig. 12.—Foreign body reaction and perivascular round cell infiltration in the vicinity of a buried hair. (Courtesy of Dr. A. Glücksmann. Reproduced by kind permission of the British Journal of Plastic Surgery).

recurrence is a likely possibility, notably in the treatment of true keloids, radiation before and after operation will most often facilitate successful surgical excision. It is strongly emphasized, however, that care must be taken to avoid such postradiation sequelae as radiodermatitis and malignancy. High doses of radiation, delivered over a short period of time, are contraindicated. Such ill-advised therapy results in radiodermatitis and local tissue changes that make subsequent surgery more difficult, and also interfere with both bony and soft tissue development in the growing child.

In our approach to this problem we have adopted the policies followed by the plastic surgery and radiotherapy services of the Presbyterian Hospital, New York. The radiotherapeutic problems involved are well illustrated in a comprehensive article by Hunter,[5] based on 20 years' experience, between 1922 and 1942. Experience with the use of radiotherapy has now accumulated over a 37-year period. One of us (J. W. McNichol) has followed this routine over a period of 18 years. The results have been gratifying and the problem of radiation dermatitis has not been encountered.

The value and importance of the team approach to this problem cannot be emphasized too strongly. As a result of consultation by the various members of the team, it may be decided that certain patients should be treated by radiotherapy only, while others should be treated by radiotherapy and operation.

Should radiotherapy alone be indicated, the following program is recommended.

One treatment is given every two weeks to a total of four doses. After a three-month period of observation, a second course of radiotherapy is administered in whole or in part depending on the patient's response and the radiotherapist's judgement.

If radiotherapy is to be combined with operation the following procedure is recommended: one course of roentgen therapy is administered as above, the area radiated including the skin to be incised if the lesion is a true keloid. Immediately after the last radiation treatment the scar is excised. As soon as the wound is healed and the sutures are removed, a second course of radiotherapy is administered.

The following radiotherapy technique is recommended by Hunter. The scar is outlined with a pencil on tracing paper, the tracing is transferred to lead foil and the latter is then cut out 3 mm. to 5 mm. beyond the outline of the scar so that a rim of normal tissue will be exposed when the lead foil is used as a shield during radiation treatment. The average radiation dose recommended is 200 r (130 Kv. p) using a 3 mm. aluminum filter. For thin scars the recommended dose is 140 r (100 Kv. p) with no filter.

Suture Marks

Lacerations which should leave relatively inconspicuous scars are often exaggerated to the point of appearing hideous, due to the formation of many transverse suture marks. The following factors influence the formation of such suture marks:

1. *Type of suture.*—Suture materials that cause irritation, such as catgut, will result in more scarring than bland sutures such as wire, nylon or silk.

2. *Size of sutures.*—The size of the suture makes very little difference if it is used

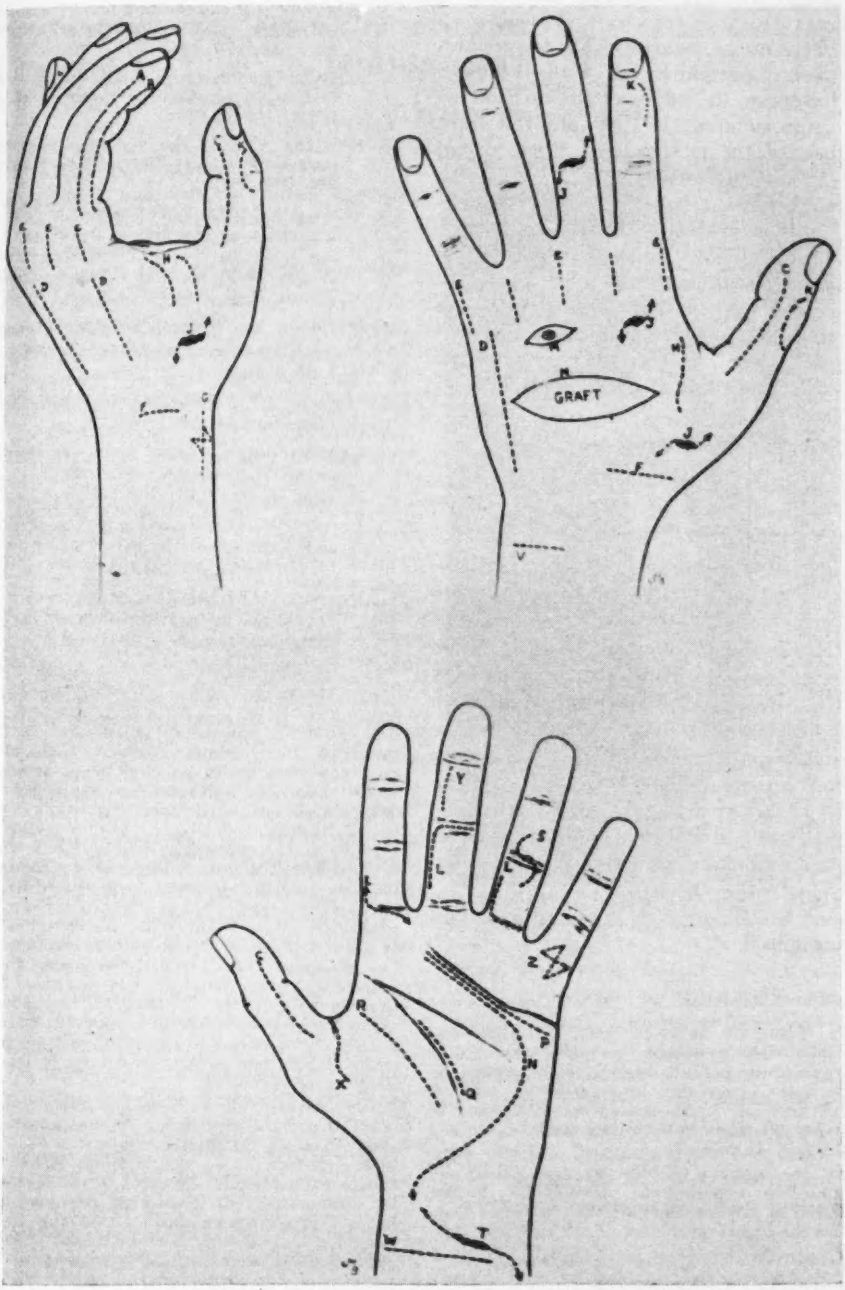


Fig. 13.—Proper incisions for surgery of the hand. (Courtesy of Dr. Julian Bruner. Reproduced by kind permission of the British Journal of Plastic Surgery).

properly, but if used improperly, the larger the stitch the larger will be the mark it leaves.

3. *Tension of sutures.*—This is probably the most important factor of all. If sutures are too loose, the edges of the incision are not approximated. If they are too tight, necrosis of the strangulated tissue occurs with resulting increase in scarring by secondary healing.

4. *Infection.*—Infection around the sutures is an obvious cause of increased scar formation at suture sites.

5. *Downgrowth of epithelium.*—This phenomenon, occurring about the suture material, may result in the formation of pits, or occasionally of skin tunnels.

Summary and Conclusions

Undesirable scars are of varying types, the most common of which is the hard, itchy, tumour-like hypertrophied scar.

The actual importance of classical Langer's lines is overemphasized and these are very difficult to remember.

Natural wrinkle lines are a much more reliable guide to good incisions and are almost always very easy to check, even while the knife is poised.

In traumatic surgery, where lacerations do not respect so-called "good lines", the surgeon should make the most of wrinkle lines where possible and rely on meticulous surgical technique to minimize scarring.

Where gross hypertrophy appears imminent a competent radiotherapist should be consulted.

Acknowledgments

Mr. Rainsford Mowlem, Mr. A. Glucksmann and their staffs generously permitted reference to their previously published fundamental works. The authors also express their appreciation to Dr. C. J. Kraissl and Dr. J. M. Brüner for permission to reproduce illustrations from their publications, and to Dr. A. F. Hunter.

We are grateful to Miss Jo Ann Ashley for assistance in preparing the photographic illustrations and to Mrs. Morski and Mrs. Margaret Watson for their secretarial help.

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Résumé

Il est de la plus haute importance d'éviter la formation de mauvaises cicatrices. Celles-ci peuvent être cataloguées en cinq types principaux: 1. Les cicatrices entravant un mouvement qui, à la tension vont se déchirer et provoquer ensuite un ulcère; de cette façon, l'augmentation progressive de tissu cicatriciel crée un cercle vicieux. 2. Les cicatrices avec tension, généralement provoquées par des incisions perpendiculaires aux lignes de Langer. 3. Les cicatrices fixées à la profondeur, dues à l'existence d'adhérences entre la peau et les parties profondes, os ou muscles. 4. Les chéloïdes vraies, surtout fréquentes chez les individus de race noire. 5. Les cicatrices hypertrophiques, qui sont de beaucoup les plus fréquemment rencontrées; on les appelle également pseudo-chéloïdes; elles succèdent aux déchirures accidentelles, aux brûlures profondes et aussi aux incisions chirurgicales.

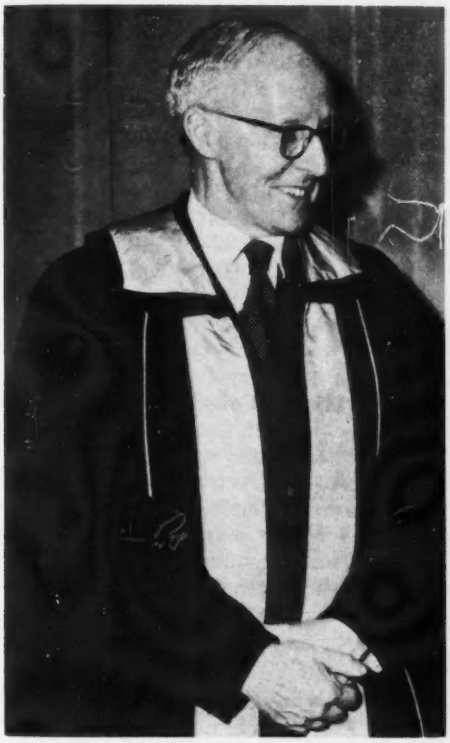
Dans la suite de l'article, les auteurs insistent sur l'importance des lignes de Langer; celles-ci sont cependant difficiles à retenir et il est souvent plus commode de se référer à un système plus simple selon les rides cutanées. Il est évident que les traumatismes ne respectent pas les "bonnes lignes": le chirurgien devra alors faire tout son possible pour réduire au minimum la surface cicatricielle en se basant sur les lignes des rides. Les cicatrices hypertrophiques pourront être avantageusement soumises à la radiothérapie; il faudra dans ce cas protéger les parties alentour par un écran de plomb soigneusement ajusté sur les bords cicatriciels.

SPECIAL COMMUNICATIONS

CANADIAN VISIT: REPORT OF THE FIRST McLAUGHLIN-GALLIE PROFESSORSHIP

C. F. W. ILLINGWORTH, M.D., *Glasgow, Scotland*

The Royal College of Physicians and Surgeons of Canada at its annual meeting in January 1960, established an Educational Endowment Fund. The College was most fortunate to receive from the McLaughlin Foundation a generous pledge for an annual grant of up to $6000 to provide for the visit of a distinguished guest teacher or investigator to one or more Canadian Medical Schools. The fund was established by Mr. R. S. McLaughlin as a tribute to the memory of the late Professor W. E. Gallie and the tour of the guest lecturer is to be known as the R. S. McLaughlin-W. E. Gallie Visiting Professorship. Professor Charles Illingworth, Regius Professor of Surgery, the University of Glasgow, was invited and consented to come to Canada as the first recipient of the lectureship. We are privileged to publish a short account of his experiences and impressions of this occasion.



Dr. C. F. W. Illingworth, Regius Professor of Surgery, University of Glasgow.

It was a great privilege to be the first R. S. McLaughlin-W. E. Gallie visiting professor, and I am glad to be offered this opportunity of giving an account of the visit, which occupied six weeks in the autumn of 1960. During this time my wife and I spent nearly two weeks in Vancouver and a similar period in Winnipeg, with short visits also to Kingston, Ottawa, Toronto and Montreal. Needless to say we met with great kindness and hospitality everywhere, renewed many old friendships and made many new ones. It is no polite fiction but sincere truth that we enjoyed every minute and were sorry when the time came to leave.

I am sure that it was a wise decision on the part of the Canadian Royal College to suggest that we should spend the greater part of the time in two centres, even though it meant missing entirely several of the Canadian medical schools and permitted only brief visits to others, for this plan enabled me to enter fully into the medical life of Vancouver and Winnipeg, to meet nearly all of the clinical teachers and most of the resident and intern staff and undergraduates. I should think that much of the benefit to be expected from the visiting Professorship must come from close contacts of this sort, and for my own part they were the most enjoyable features of my tour.

I should like to express my thanks to those who were responsible for arranging our programs, particularly Dr. Allan McKenzie and Dr. Colin Ferguson and the members of their departments. I know well

what a great deal of trouble is involved in planning a timetable of this sort, involving lecture times, meetings, visits to other hospitals, contacts with many colleagues, and so on, and to superimpose this upon routine hospital duties.

The program they arranged was indeed a kind of royal progress, with a minute by minute schedule of visits, interviews, lectures and off-the-cuff commentaries, and an occasional brief interlude for refreshment or reflection. Altogether, it was quite arduous but immensely stimulating.

It has been suggested that I should take this opportunity of giving my impressions of surgery in Canada as compared with that in my own school. I am well aware of the fallacies of an impression gained in a brief visit. The fact that I was involved in, on the average, three seminars or teaching rounds or conferences a day, and was invited to a social party seven evenings a week, testifies to Canadian hospitality but not to the normal Canadian way of life. The fact that every operation I saw performed was for valvotomy or a septal defect does not imply that Canadian surgeons never treat hernia or gallstones.

The conditions under which surgery is practised in the two countries are also so different that it is hazardous although tempting to make comparisons. In Glasgow and indeed in the United Kingdom generally, people of all social classes come into the ordinary wards of the main teaching hospitals, making the best of the rather primitive conditions and the lack of amenities which still characterize our outdated hospitals, and as a rule they are content to receive treatment, whether at the hands of staff men or registrars or interns, on the general expectation of getting the best treatment appropriate to their particular needs. In Canada, on the other hand, patients are accustomed to the better conditions available in your more modern hospitals and they prefer greater privacy, while as to their medical attention, it is my impression that they prefer to select and pay their own surgeons or physicians and, as a corollary, expect them to give their detailed personal attention throughout the whole course of the treatment.

It seems probable that these differences will become more marked with the passage of time. In the U.K. there is an increasing tendency for patients who formerly paid for treatment in our admittedly inefficient nursing homes to seek admission to the ordinary wards of the main hospitals. In Canada the opposite tendency is evident, at least in some centres, where patients who hitherto would have been treated in the staff wards tend more and more to be regarded as semiprivate or private cases. This trend, if it continues, will obviously have repercussions on the facilities for medical education, as indeed is already beginning to be evident in some of the schools.

Nevertheless, despite these contrasts, I was more impressed by the similarities than by the differences between my own school in Glasgow and the Canadian centres I visited. Fundamentally our attitude towards our patients and our approach to treatment are much the same, while our views on teaching and research are not greatly different. We in the U.K. like you in Canada, tend to be rather conservative in assessing new methods of treatment. In our diagnosis and ward care, while making full use of laboratory techniques, we, like you, also appreciate the value of clinical judgment and experience. In our research laboratories we are similarly concerned with basic problems as well as with operation techniques.

The problems of current interest to surgeons are much the same in the two countries and I was interested to see how close our thinking is, even on matters which are in a state of flux and change. For example, in breast cancer, the high failure rate of orthodox methods of treatment is now universally acknowledged and there is general agreement that the endocrine ablations, while they give gratifying remissions in a proportion of cases, are less beneficial than our earlier optimism led us to expect. In both countries at the present time active research is going on to explore the potentialities of the antimitotic and cytotoxic agents though with little immediate prospect of success.

It is true that there are some minor differences in surgical problems in the two countries. In my visits to Canada and also

to the United States I am always impressed with the frequency with which chronic pancreatitis comes up as a subject of discussion, whereas in the U.K. we regard it as something of a rarity. We can probably disregard flippant conjecture as to the relative etiological significance of rye and scotch but if there is any real difference in the incidence it would be very interesting to explore the subject further. Conversely perforated peptic ulcer is not often mentioned in Canada whereas in the U.K it is an ever present problem.

The problem of wound sepsis is certainly a very topical one in both countries. This is not a new problem and I do not think it is increasing. Almost certainly there was much more wound sepsis formerly than at the present time but in those days it was accepted as an inevitable feature of surgical work whereas now we are more critical and with better bacteriological services we can estimate its frequency and trace its routes of spread more accurately. In Canada, as in the U.K., the problem is being tackled vigorously and I was much impressed by the energetic measures being taken in one or two Canadian centres to control its incidence.

One of the most enjoyable features of my visit was provided by the frequent opportunities to take part in undergraduate teaching and graduate training programs. It is difficult to compare your educational methods with our own as the basic conditions are so diverse. I think it is probably still true on the whole that in the U.K. generally the teaching is more didactic, with more formal lectures, more note taking and more written examinations. However, there are some exceptions to this generalization and my own unit in particular follows Canadian practice more closely in this respect. Consequently I felt completely at home in the teaching rounds in Vancouver, in Winnipeg and the other cities I visited and I enjoyed the contacts with the students enormously.

Graduate teaching, contrariwise, tends to be more fully organized in Canada than in the U.K. where only a few hospitals such as my own have so far instituted a regular "Registrar rotation" service, and so far as I know there is nothing on this side at all comparable, for example, to the very thorough graduate training scheme which I saw in Winnipeg.

At this point I should like to take the opportunity of commenting on the work carried out by the residents and interns of the staff wards of the centres I visited. It seemed to me that they were carrying out their responsible duties with great assiduity and ability. I had few opportunities of comparing the standard of treatment given to private patients but I find it difficult to believe that it could be any better. In the centres I visited the resident work was well supervised and while the individual responsibility given to residents was greater than is usual on this side I got the impression that the men chosen for these posts were well able to carry the extra load of responsibility. Certainly I am in no doubt that with proper supervision a good teaching unit in Canada as in the U.K. offers the best facilities for good treatment.

It was very pleasant in the course of our tour to have the opportunity to visit the fine new building of the Royal College in Ottawa, where Dr. Graham and his staff made us very welcome, and where I had the privilege of transmitting a message of greeting, and a token of friendship in the shape of a gavel, from our own ancient corporation, the Royal Faculty of Physicians and Surgeons of Glasgow. Note that these two bodies include within their members both physicians and surgeons and indeed practitioners of every branch of medicine. I am sure this is a good plan. Medical care is indivisible and the division between physicians and surgeons is quite artificial. Certainly there is a great deal in common between general surgeons and internists, between cardiologists and those who perform heart operations, between neurosurgeons and neurologists. In Glasgow the bonds are becoming closer rather than laxer, as is shown in our joint "grand rounds" and research seminars, and I am interested to see the same tendency developing in Canada.

One of the last engagements on my tour was a dinner party at the house of Mr. Sam McLaughlin, whose name is associated with the late W. E. Gallie in the

Visiting Professorship which I held. It was a very pleasant occasion, and particularly delightful to see that a great industrialist can still take such a personal interest in medical affairs, and even in the doings of every individual Fellow supported by his Trust. I had met many ex-Fellows in the course of my tour and there is no doubt of the value which the Trust is giving, through them, to Canadian surgery in general. I hope that in the future more of them will elect, in their year as McLaughlin Fellows, to visit my unit in Glasgow, where they can be assured of a warm welcome as well as, I believe, a profitable experience in clinical work or research.

Résumé

Le Dr. C. F. W. Illingworth est le premier récipiendaire de la bourse McLaughlin-Gallie. Cette nouvelle fondation se propose de permettre l'organisation de visites au Canada de professeurs étrangers éminents. Le Professeur Charles Illingworth est professeur de Chirurgie à l'Université de Glasgow.

Après avoir remercié les organisateurs de son voyage, le Dr. Illingworth donne ses impressions sur la chirurgie au Canada. Il souligne que, d'une façon générale, les hôpitaux de Glasgow, plus anciens que les hôpitaux canadiens, se trouvent être moins confortables; le malade canadien est certainement habitué à plus de modernisme et il se fait plus volontiers traiter en privé. Ces différences iront sans doute en augmentant; ceci risque évidemment d'avoir des répercussions sur les possibilités d'enseignement, de par la diminution progressive des cas publics. Il y a par contre peu de différences entre les médecines canadiennes et anglaises en ce qui concerne l'attitude du médecin vis-à-vis du malade et l'organisation des traitements. Ici et là, on utilise de la même façon les techniques de laboratoire.

Les problèmes scientifiques qui intéressent le plus les chirurgiens des deux pays sont les mêmes: dans la lutte contre le cancer, tout le monde travaille actuellement sur les drogues antimitotiques. Il est curieux de noter une incidence et une fréquence beaucoup plus forte de pancréatites chroniques sur le continent américain.

En ce qui concerne l'enseignement, gradué ou pré-gradué, l'auteur pense que les méthodes utilisées en Angleterre sont plus conservatrices: il y a plus de cours ex cathedra, plus de notes à prendre et plus d'examens. L'enseignement post-gradué est plus developpé ici.

ROSE AND CARLESS MANUAL OF SURGERY. Consulting Editor Sir Cecil Wakeley, Bt., K.B.E., C.B. Edited by Michael Harmer and Selwyn Taylor, assisted by 15 contributors. 19th edition. 1389 pp. Illust. Ballière, Tindall and Cox, London, 1960. $14.25.

For over 60 years undergraduate students as well as graduates studying for their Fellowship have made use of this book in their studies. It has generally been considered to contain more information than any comparable textbook in surgery.

It is a particularly comprehensive book as it has been in the past. It is different from most general surgical textbooks in that it includes sections on ophthalmology, otolaryngology, gynecology and tropical surgery.

The present edition has been published in one volume and this has many obvious advantages. The deletion of the chapter on anesthesia will not detract from it, inasmuch as there are now many textbooks in anesthesia which may be consulted. Another pleasing feature has been the incorporation of a brief biography of the two original authors. This will meet with the approval of those who have an interest in the history of surgery.

The first chapter deals with the general principles related to infection, shock, electrolyte disturbances, wounds, burns, chemotherapy, blood transfusions and similar topics. It has been brought completely up to date with the latest information concerning advances in the care of the surgical patient.

Subsequent chapters are devoted to the surgery of the different organs and systems. Each subject is dealt with in an orderly fashion, first outlining the pathology or altered anatomy, signs and symptoms, diagnosis and treatment. Detailed discussion of the techniques of operations is not provided but the general principles are outlined.

It is a great tribute to any book that it is still in use 60 years after publication of the first edition. With this new edition, Rose and Carless will be assured of a permanent place in all medical libraries and reading rooms. It is one of the most up to date and complete works in surgery. It will be particularly helpful to medical students, practising surgeons and any who wish to consult an excellent surgical textbook.

TRIBUTE TO THE MEMORY OF SIR GORDON GORDON-TAYLOR, K.B.E., C.B., M.D., Hon. LL.D., Hon. Sc.D., M.S., F.R.C.S., Hon. F.R.C.S.(Ed.), Hon. F.R.C.S.(I.), Hon. F.R.C.S.[C], Hon. F.F.A.R.C.S.

JOHN LAING McDONALD, M.B., F.R.C.S.[C], *Toronto*

IT IS WITH a feeling of deep humility that I perform this labour of love by attempting to pay my personal tribute to this great man whom I loved and admired so much. "G.T.", as he was affectionately known by his students and house surgeons, was a brilliant classical scholar and a master of the English language. His many surgical contributions, published in various medical and surgical journals throughout the world, and his numerous tributes paid to departed friends and colleagues, were written with a phraseology and beautiful choice of words which were like soft music to the highly sensitive ear.

Already many fine tributes to Sir Gordon Gordon-Taylor have been written by his former students and house surgeons. These



SIR GORDON GORDON-TAYLOR

The above is a photograph of a recent portrait by JAMES GUNN, R.A., commissioned by the Australasian College of Surgeons and now hung in the home of the College in Australia.

I wish to acknowledge my deep appreciation to the artist for granting permission to publish it in the *Canadian Journal of Surgery*. J.L.McD.

have given a sketch of the early life, academic achievements and the many honours that have been conferred on this brilliant man. I shall attempt to review these but briefly, and to give more time to the man, as viewed through the eyes of a Canadian.

Sir Gordon was born in Aberdeen, Scotland, on March 18, 1878. He was educated at Gordon's College and Aberdeen University where he studied Classics and obtained his M.A. degree with honours in 1898. He was fortunate in having the privilege of obtaining his early training in the University of Aberdeen, one of the greatest centres of learning in the English-speaking world. His later life proved that he must have been one of the most brilliant graduates of that institution.

With the loss of his father early in life, he accompanied his widowed mother and other members of his family to London, where he became a student in medicine at the Middlesex Hospital, from which institution he obtained his M.B., in 1903. From the beginning of his medical career he showed a keen interest in anatomy, winning the medal in anatomy at the time of taking his intermediate examination. With a fellow student and personal friend, Victor Bonney, he then determined to further his knowledge of anatomy by working for the degree of B.Sc. This demanded many hours of labour, well into the night, and both men succeeded in obtaining their B.Sc. in Anatomy with honours. In 1906 he obtained his M.S. from the University of London, and in the same year he became a Fellow of the Royal College of Surgeons of England.

Sir Gordon was appointed to the surgical staff of Middlesex Hospital on the service of Sir John Bland-Sutton in 1908. In 1921 he succeeded Sir John as head of the service and about that time he became Chief Surgeon to the Middlesex Hospital.

During the First World War he served as a surgeon with the R.A.M.C., during which period he made many valuable contributions to the field of military surgery, performing many operations in field ambulances and casualty clearing stations, saving the lives of many severely wounded young men. Sir Gordon's greatest contributions, I believe, during that period, were in the treatment of gunshot wounds of the abdomen. While serving close to the Canadian lines, he met the late Dr. Bruce Robertson who was the first man to successfully carry out blood transfusions to combat shock and loss of blood in the severely wounded. In many of the medical histories written about the First World War, it has been stated that blood transfusions were introduced in the treatment of severely wounded in warfare by the Americans. Sir Gordon was one who stated that blood transfusions were being performed in France in the winter of 1915-1916 by the late Bruce Robertson, long before the Americans had entered the War. Sir Gordon retired from war service with the rank of Major, and was awarded the Order of the British Empire.

Soon after his appointment as Chief of the Service at the Middlesex Hospital, it became the most sought-after service for the surgical training of the undergraduate students, and for the house officers competing for training in surgery. It was my good fortune to be appointed as a house surgeon to G.T. in the winter of 1921-22. This, I believe, was only possible because the most brilliant Middlesex graduate, Dr. Bedford, chose to become a physician, and later became the most outstanding cardiologist of Great Britain. The fact that my father was born in Scotland, within 60 miles of the birthplace of Sir Gordon, also may have proved helpful.

Many pages could be written about the cherished memories I retain of the period spent with G.T., which was one of the most pleasant experiences of my life. During that period, he established his own operating theatre in close proximity to his surgical wards. This made it possible for him to arrange his operating lists as and when he wished. He had regular operating days on Monday and Friday afternoons, using Wednesday afternoon for what he called "clean-up" work.

The nurse in charge of his service, and also the supervisor of his operating room, was Sister Waterman, who was a truly remarkable character. She had served as a Nursing Sister throughout the First World War, and was a plump, jolly soul,

with a rare sense of humour, and I can still recall her hearty laughter. She was devoted to G.T. and she seemed to carry out her heavy responsibilities of efficiently supervising the operating room as well as the Wards, with apparent ease. The operating lists of Monday and Friday were long, and included many of the most major operations in surgery of that day, including thyroidectomies, radical operations on the mouth and neck for cancer, radical mastectomies, partial gastrectomies, and so on, with several attempts at excision of the œsophagus for malignancy. These operations went smoothly, without apparent confusion, and at an appropriate hour in the afternoon we would retire to Miss Waterman's office for tea, to which any visiting surgeons of that day were invited.

Sir Gordon firmly believed in the value of the apprenticeship method of learning surgery, and it was his custom to have his house surgeon perform all the standard operations in surgery under his supervision. He would perform one or more of the various operative procedures with his house surgeon assisting, and as time went on he would reverse the procedure, assisting his house surgeon in similar operations. I feel sure that none of us will ever forget the wonderful training we obtained from G.T. nor the many things we learned about surgery during the tea hour, and at other periods when not actually working in the operating room.

It was the custom of Sir Gordon, whenever his operating list carried one beyond the normal hour of dinner for the house staff – which occurred about once a week –, to say as we left the operating room, "I would like you to hurry, change your clothes and meet me at –", his favourite restaurant. On arrival, a dry Martini would be waiting, and seated at the table would be Lady Gordon-Taylor, a most charming person, with her two sisters. Following dinner, we would move off to G.T.'s favourite place for dancing, where we would enjoy an evening of ballroom dancing, until about 11:30 p.m. Ballroom dancing, I believe, was G.T.'s favourite form of relaxation, and he was a beautiful dancer..

G.T. never owned nor drove a motor car. However, during his active surgical career he always had a Daimler car, with the same chauffeur, on lease from the Daimler Company. He was very fond of walking, and preferred to walk from his home or consulting rooms to and from the Middlesex. He did, however, use the Daimler when travelling to the various nursing homes in London, when performing operations on his private patients. On these occasions he was always accompanied by his house surgeon, who assisted him with the operation, and who always received an envelope with a generous fee enclosed, at the completion of the operation.

Sir Gordon was an examiner in surgery at the University of Leeds, and was a great admirer of Sir Berkeley Moynihan, later Lord Moynihan. During G.T.'s absence from London, while examining students in Leeds, attending meetings, and performing other duties, he left the care of his patients both at the Middlesex and in the various nursing homes, to his house surgeon. I well remember what a thrill it was to be picked up by the Daimler, with the favourite driver, and to make a tour of the various nursing homes in London, which were at that time supported in large measure by G.T. and his patients.

It was Sir Gordon's usual custom to invite his house surgeon, during his period of service, to accompany him as his guest on a visit to Sir Berkeley Moynihan, Surgeon-in-Chief of Leeds Royal Infirmary. There, an operative program would have been arranged previously by Sir Berkeley, and it was a great thrill for me to see that master surgeon in action, and to visit the pathological laboratory closely adjacent to the operating suite, to hear a learned discourse on the pathological material that had just been removed from the previous patient. Such was the modesty of G.T. that, I well remember, on that visit to Leeds, I was introduced to Sir Berkeley as a former house surgeon of Dr. H. A. Bruce, and not as his (G.T.'s) house surgeon.

I recall with pleasure another custom then observed at the Middlesex regarding the relationship of the house surgeon to his chief. It was an unwritten law that the house surgeon meet his chief at the entrance to the hospital. There one would observe G.T. approaching, wearing his

favourite tweed suit with a pink carnation in the buttonhole, walking briskly with his hat at a jaunty angle, complete with cane and gloves. Even in cold weather, he rarely donned a topcoat, and on those occasions, he would carefully move the pink carnation from his topcoat to his double-breasted jacket. After I had deposited his hat, cane and gloves in the cloak-room close by, we would then proceed to the wards or the operating floor. After completion of his duties in the hospital we would return to the entrance where I would return to him his hat, cane and gloves. To me there was a rare charm and dignity associated with this ritual, and I believe we would do well to emulate, in this country, some of the formalities prevailing in the hospitals of London. It would do much to add to the respect and dignity of the medical profession.

Early in his surgical career, Sir Gordon became an examiner in anatomy in the Primary Fellowship Examination of the Royal College of Surgeons, and he continued in this capacity throughout the remainder of his life, in his later years serving as an alternate examiner when for any reason the examiner appointed was unable to be present. I recall G.T. examining me in anatomy in the autumn of 1920. I do not believe one can truthfully say that he looks back with pleasure on any examination, but I well remember G.T.'s courtesy to the students, and the charming way with which he succeeded in putting them at ease.

In 1924 the late Dr. W. E. Gallie gave the Hunterian Lecture at the Royal College of Surgeons of London. He was accompanied on his trip to England by his old friend, the late Dr. D. E. Robertson. Some time before they left Canada I had written G.T. with the request that he put on an operative program at the Middlesex during Dr. Gallie's stay in London. This was the first occasion on which these two outstanding surgeons met, and it was the beginning of a friendship which lasted throughout the remainder of their lives. It was followed by a closer relationship between Middlesex and Toronto, eventually leading to an exchange of professors between these two great medical centres. The present arrangement was worked out between Dean Sir Brian Windeyer of Middlesex, and our Dean, Dr. J. A. MacFarlane of Toronto. This exchange of professors commenced in 1956, and I do hope that it will continue indefinitely.

In 1939 Sir Gordon made his first visit to Toronto to give the Lister Oration to our students of medicine on April 5, the birthday of Lord Lister. This so-called Balfour Lecture was donated several years previously, by Dr. Donald Balfour of the Mayo Clinic, as a gift to his alma mater.

On the occasion of G.T.'s visit to Toronto where we had the pleasure of his company for a period of about one week, he lived as a guest at the York Club, and such was the nature of the man that on each subsequent Christmas, the York Club received a card from Sir Gordon.

It so happened that his visit coincided with the meeting of the Canadian Association of Clinical Surgeons, and part of the programme consisted of a series of operations performed by various Toronto surgeons on the operating floor of the Toronto General Hospital. While driving from the York Club to the Toronto General on the morning of this meeting, G.T. turned to me and said—"Tell me, who is the best surgeon in Toronto?" After some hesitation I stated that we had many good surgeons, and that I thought it would be difficult for me to answer his question directly. However, I said, if you would like me to give you the name of the surgeon that I would like to have operate on me, I would choose Dr. Norman Shenstone. He said "Why?" I replied that in my opinion Norman Shenstone was not only a good surgeon but a man of wide experience and sound judgment and I felt that I would be reasonably safe in his hands. G.T. remarked that he would like to see Dr. Shenstone at work. It was fortunate that among the operations booked for that morning was a total pneumonectomy to be performed by Dr. Shenstone on a patient, who, I believe, suffered from a cancer of the lung. Dr. H. J. Shields was giving the anæsthetic. The operation had proceeded to the point where the chest was opened and the stage was set for the removal of the lung. Before continuing further, Dr. Shenstone noted that the blood

from the incision was a little blue. He turned to the anæsthetist and said—"Harry, are you in trouble?" Dr. Shields stated that he did not like the condition of the patient. Dr. Shenstone then turned to Sir Gordon and said "I am very sorry, sir, that it will be necessary for me to complete this operation on another day," and with that he proceeded to close the chest. As we left the operating room, G.T., with the usual twinkle in his eye, and a slight twitch of the left side of his forehead, turned to me and said—"You know, John, I believe I would like to have that man operate on me."

Sir Gordon was then Vice-President of the Royal College of Surgeons of England, and within a few months an invitation was extended to Dr. Shenstone, from the Royal College, to accept the degree of Honorary Fellow of the Royal College of Surgeons of England. No one more justly deserved that honour than that modest man, Dr. Norman Shenstone, who has done so much pioneer work in the surgery of the chest, and his many friends were delighted that he should be so honoured by the Royal College of England.

On the occasion of Sir Gordon's visit to Toronto he stated that during his travels throughout the Commonwealth, it gave him a great deal of pleasure to have one of his former house surgeons perform an operation in his presence. Following this request I arranged to perform a lumbar sympathectomy at the Hospital for Sick Children, on a little child, a victim of infantile paralysis with some shortening of the leg associated with coldness of the extremity on the affected side. Fortunately this operation went very well, and as is usual under the circumstances, it appeared to be a very simple surgical procedure. I apologized to G.T. for not being able to perform an operation that might be of greater interest to him. As was so characteristic of the man who always tried to encourage his younger colleagues, his reply was — "But, my dear fellow, I am interested in all branches of surgery. The operation was performed by you in a superb manner," and then he proceeded to tell me about the unfortunate experience of another surgeon whom he had watched doing a similar operation.

During the second World War, Sir Gordon, though then a man well past 60 years of age, again came to the service of his country, and after being turned down by the R.A.M.C. becaue of his age, was very soon accepted in the senior service, serving as Surgeon Rear-Admiral in the Royal Navy throughout the remainder of the war. While serving with the Royal Navy he travelled extensively throughout Great Britain and made many trips to the United States, Canada, and to other countries of the Commonwealth. In 1943 he travelled to Russia for the British Council, and during that visit, on behalf of the Royal College of Surgeons of England, he conferred the Honorary Fellowship of the College on the famous Russian surgeons, Judin and Burdenko. In recognition of his distinguished services during World War II, he was rewarded by his sovereign, the late King George VI, by being made a Companion of the Most Honourable Order of the Bath and receiving a Knighthood in the Most Excellent Order of the British Empire. He was also named a Commander of the United States Legion of Merit.

Following the war, Sir Gordon continued to hold an active interest in surgery, devoting most of his time to consulting practice and writing. He was appointed Official Adviser to Overseas Students from the Commonwealth by the Royal College of Surgeons. In that capacity he spent several hours, two mornings each week, at the home of the Royal College on Lincoln's Inn Fields Road. There he would meet postgraduate students from all parts of the Commonwealth, discuss with them their plans for further training, and greatly assist them in obtaining suitable appointments for such training, in all parts of Great Britain. Frequently following these interviews he would invite them to have lunch with him at the Ritz Hotel on Piccadilly, where, in the dining-room, his favourite table, overlooking Green Park, would be waiting for him. Many of the staff of the Ritz, and even their fathers before them, had been former patients of Sir Gordon. He always received the loving

attention and devoted service which he so justly deserved.

As a result of his extensive travels throughout the Commonwealth and his meeting with so many young men and women at the Royal College, he became by far the best known and most highly regarded British surgeon of all time throughout the British Commonwealth. He followed with keen interest the progress of his younger colleagues, and he appeared to be as pleased with their attainments as would be a father with his son.

Sir Gordon had a remarkable memory for names, and his numerous friends and colleagues throughout the Commonwealth were always remembered with a card from him at Christmas. These cards were always addressed in his own hand, together with a suitable personal note, and were ready for mailing about August. I believe it was his custom to send about 1000 Christmas cards each year. Some 800 cards were already addressed and ready for mailing before G.T.'s death last year. It was with a feeling of sadness that one received his card some time after his death, sent by his faithful secretary, Miss Johnson.

Sir Gordon gave many lectures and addresses on surgery and other subjects throughout Great Britain, the various countries of the Commonwealth, the United States and many other countries. These were all most carefully prepared, written and rewritten until they had reached the perfection in composition and language that he would accept, and then so carefully memorized that they were delivered by him without a note.

Sir Gordon was the recipient of numerous honours from various centres of learning in Great Britain, the various countries of the Commonwealth, the United States and other nations. These have already been recorded elsewhere and I will not, at this time, make any attempt to review the lengthy list of brilliant addresses and the honours bestowed upon him.

Sir Gordon was devoted to his mother, a Gordon, and a member of that famous Scottish clan who played such a prominent part in the early history of Scotland and who later distinguished themselves in the service of Great Britain and the Commonwealth over a period of more than 200 years. G.T. was proud to be a member of the Gordon clan, as one can assume from the name he chose. He was a brilliant example of the true Scot, generous, kindly and possessing an unusual capacity for making friends and giving something of himself to them.

Sir Gordon's interest in cricket started when he was a boy at school and he retained a keen interest in the game throughout his life. He was a member of Lord's Cricket Ground, and especially during his latter years he frequently would be found at Lord's watching a test match. It was while crossing the road near the entrance to Lord's Cricket Ground that he was struck by a motor car, sustaining serious injuries which led to his death a few hours later, on September 3, 1960, in his 83rd year.

With the death of Sir Gordon Gordon-Taylor the world of medicine lost its greatest classical scholar and most distinguished surgeon, and the British Commonwealth of Nations lost their greatest ambassador of good will. We, his former students and house surgeons throughout the Commonwealth, have lost an old friend whom we loved and admired, and who will remain always a guiding light in our lives.

Our sympathy goes out to his only living sister, and to Miss Johnson, his devoted and faithful secretary for so many years.

Résumé

Sir Gordon Gordon-Taylor est né à Aberdeen, en Ecosse, en 1878. Il fit ses études au Gordon's College puis à l'Université d'Aberdeen, où il obtint avec distinction son diplôme de M.A.

Alors qu'il était encore jeune homme, son père mourut et, de ce fait toute sa famille dut se déplacer et habiter Londres. Il s'inscrivit comme étudiant en médecine au "Middlesex Hospital". Cette institution lui décerna en 1903 le grade de bachelier en médecine. Durant ces études il montra un interêt tout particulier pour l'anatomie et ayant gagné une médaille pour ses connaissances dans cette discipline, il décida d'approfondir cette science et commença son baccalauréat es sciences. Il obtint en 1906 son M.S., et en même temps devint "Fellow" du Collège Royal de Chirurgie d'Angleterre.

Il fut nommé membre du bureau médical de l'Hôpital du Middlesex dans le service de Sir John Bland-Sutton en 1908. En 1921, il succéda à Sir John comme directeur du service et peu après devint Chirurgien-Chef de l'Hôpital.

Pendant la première guerre mondiale, il servit dans le corps expéditionnaire britannique comme chirurgien. Il contribua grandement au progrès de la chirurgie de guerre, opérant souvent dans les ambulances avancées, sauvant les vies de nombreux soldats; sans aucun doute, l'un de ses plus grands mérites fut l'amélioration du traitement des blessures de l'abdomen par balles. Il eut l'occasion de faire la connaissance de Bruce Robertson, un chirurgien canadien grandement intéressé dans les questions de transfusion sanguine: Sir Gordon coopéra avec lui et adopta ses idées: de sorte que, contrairement à ce que l'on croit généralement, ce sont ces deux hommes qui ont répandu l'usage de la transfusion d'urgence.

A la fin de la guerre, Sir Gordon fut nommé Major et reçut la médaille de l'Ordre de l'Empire Britannique.

Il reprit alors son poste de Chirurgien-Chef de l'Hôpital du Middlesex. Il réorganisa l'enseignement des internes et des résidents, en s'attachant surtout à la pratique. Les séances opératoires étaient le lundi et le vendredi dans l'après-midi.

Sir Gordon entretenait des relations scientifiques très étroites avec Sir Berkeley Moynihan qui devint plus tard Lord Moynihan, pour lequel il avait une très grande admiration.

En 1939, Sir Gordon fit un voyage au Canada et y donna une série de conférences.

Lorsque la deuxième guerre mondiale éclata, bien qu'il eut atteint la soixantaine, Sir Gordon tint à rejoindre l'armée; il occupa l'emploi de Vice-Amiral Chirurgien dans la marine: ce fut pour lui l'occasion de nombreux voyages à travers l'Angleterre, le Canada, les Etats-Unis et les pays du "Commonwealth". Sa Majesté le Roi Georges VI le fit Compagnon de l'Ordre du Bain et Chevalier de l'Ordre de l'Empire Britannique. Il fut aussi nommé Commandeur de la Légion du Mérite des Etats-Unis.

Il consacra la fin de sa vie à l'enseignement, à la lecture et à la publication de divers ouvrages. Il mourut, d'accident, à l'âge de 83 ans.

Le monde médical a perdu un de ses plus grands chirurgiens, et l'un des plus distingués.

The *Canadian Journal of Surgery* is published quarterly by Canadian Medical Association. Subscription rates are $10.00 a year or $2.50 for individual issues. Yearly subscriptions and back issues are available on request from the Canadian Journal of Surgery, C.M.A. House, 150 St. George Street, Toronto 5, Ontario.

CANADIAN JOURNAL OF SURGERY

All communications concerning this Journal should be marked "Canadian Journal of Surgery" and addressed to the Editor, C.M.A. Publications, at C.M.A. House, 150 St. George St., Toronto 5.

The Journal is published quarterly. Subscription is $10 per year, and starts with the January 1 issue of each year. (It would be greatly appreciated if subscribers would please add bank exchange to their cheques.)

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Manuscripts of original articles, case reports, and other contributions should be forwarded with a covering letter requesting consideration for publication in the *Canadian Journal of Surgery*. Acceptance is subject to the understanding that they are submitted solely to this Journal, and will not be reprinted without the consent of the author and the publishers. Acceptance or rejection of contributions will be determined by the Editorial Board. As space is available, a limited number of case reports will be published. Articles should be typed on one side only of unruled paper, double-spaced, and with wide margins. Carbon copies cannot be accepted. The author should always retain a carbon copy of material submitted. Every article should contain a summary of the contents. The Concise Oxford Dictionary will be followed for spelling. Dorland's American Medical Dictionary will be followed for scientific terminology. The Editorial Board reserves the right to make the usual editorial changes in manuscripts, including such changes as are necessary to ensure correctness of grammar and spelling, clarification of obscurities or conformity with the style of the *Canadian Journal of Surgery*. In no case will major changes be made without prior consultation with the author. Authors will receive galley proofs of articles before publication, and are asked to confine alterations of such proofs to a minimum.

Reprints

Reprints may be ordered on a form which will be supplied with galley proofs. It is important to order these before publication of the article, otherwise an extra charge for additional type-setting will be made.

References

References should be referred to by numerals in the text and should be set out in accordance with the *Cumulative Index Medicus* abbreviation of journal name and general style. They should include in order: the author's name and initials in capitals; title of the article; abbreviated journal name; volume number, page number and year. References to books should include in order: author's name; title of book; title of publishing house; city of publication; number of edition (e.g., 2nd ed.); year of publication.

Illustrations

A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10″ x 8″. Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

Language

It should be clearly understood that contributors are at full liberty to submit articles in either English or French, as they please. Acceptance will be quite independent of the language of submission. If the contributor wishes, he may submit an informative summary of not more than 300 words in the language other than that in which he has submitted the article. For example, an article in English must carry an English summary and may, if the author wishes, carry a more detailed summary in French.

The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

Two events of interest to Fellows of the Royal College took place at the Annual Meeting in Ottawa in January of this year. The first evening of the meeting saw the new headquarters building officially opened by the Prime Minister of Canada, the Right Honourable John Diefenbaker, in a colourful ceremony. This was witnessed by a large group of attending Fellows and their wives, through the medium of closed circuit television.

At Convocation held on the second evening of the meeting, an honorary Fellowship was conferred on Major-General Georges Vanier, Governor-General of Canada. At the same time an Honorary Fellowship was given to Sir Walter Mercer, emeritus professor of Orthopedic Surgery of the University of Edinburgh for his outstanding work as a teacher in his field. An Honorary Fellowship was also granted *in absentia* to Mr. Samuel McLaughlin for his generous contributions to the cause of medicine in Canada.

Registration at the Royal College Regional Meeting held in London, Ontario, in November 1960 exceeded 300 and the meeting proved to be a great success; again auguring well for the future. At present, plans are being worked out for a Western Regional meeting in Saskatchewan next Fall.

It was announced recently that the Sims Commonwealth Travelling Professor to Canada for 1961 will be the outstanding and well known cardiologist, Dr. Paul Wood, Physician to the National Heart Hospital, London, England.

The McLaughlin-Gallie Travelling Professor for 1961 is to be Sir George Pickering, Regius Professor of Medicine at the University of Oxford. Both Professors will visit Canada in the Fall of this year. Details of their itineraries in Canada will be announced later.

The results of this year's Fellowship examinations have been announced in detail previously. The overall percentage of successful candidates in surgery and the surgical specialties for this year was 32.8% as compared with 40% for 1959. This may be compared with the results in Certification for 1960 of 58% and 1959 of 60%.

At the recent meeting of Council specific recommendations from the Credentials Committee were discussed concerning the establishment of a Fellowship examination modified for Cardiovascular and Respiratory Surgery, this to replace the present Certification examination in Thoracic Surgery. Further details concerning the Regulations and Requirements of graduate training relating to this examination will be forthcoming in the future.

Recently adopted statements of Council policy on dichotomy and on itinerant surgery will be found elsewhere in this issue of the *Canadian Journal of Surgery*.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary
February 27, 1961.

RESOLUTION AND STATEMENT PASSED BY THE COUNCIL AT A MEETING IN OTTAWA

January 17, 1961

WHEREAS this Council of The Royal College of Physicians and Surgeons of Canada has considered the problem of ITINERANT SURGERY, that is, surgery performed on a patient unfamiliar to the surgeon at a distance from his practice and where the diagnosis, pre and postoperative care is delegated to another physician, and whereas this Council of The Royal College of Physicians and Surgeons of Canada recognizes that the operative procedure is only a portion of the surgical care of the patient,

BE IT RESOLVED that this Council of The Royal College of Physicians and Surgeons of Canada condemns ITINERANT SURGERY *as defined above* as

being detrimental to the best interests of the patient and contradictory to the present concepts of comprehensive surgical care.

When the circumstances are such that it is in the best interest of a patient's care to have an operative procedure carried out by a surgeon in a hospital remote from the site of his regular practice, that surgeon must be responsible for the diagnosis, the pre and postoperative care of the patient.

The surgeon's fee for these services must be commensurate with the services rendered and for the time that he has been away from his regular place of practice.

In view of the foregoing it becomes evident that any attempt on the part of practitioners or prepaid medical plans to divide the regular surgical fee between the participating surgeon and the practitioner in this situation is not ethical or in the best interests of the patient.

AN INFORMATORY STATEMENT REGARDING THE POSITION THE ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA TAKES TOWARD DICHOTOMY OF FEES, AND THE PRINCIPLES OF FINANCIAL RELATIONS IN THE PROFESSIONAL CARE OF THE PATIENT

(Reference may also be made to the Code of Ethics as set up in Article 12 of the By-Laws of the Royal College of Physicians and Surgeons of Canada)

1. IN HIS RELATIONSHIP WITH THE PATIENT, a Fellow of The Royal College of Physicians and Surgeons of Canada or a Certificant of the College should inform the patient, or a member of his family, of the identities of the doctors who will collaborate in the care of the patient; should be willing to discuss his fee with the patient or a member of the patient's family, prior to submitting a statement; and should present a statement for his own services to the patient.

(a) INFORM THE PATIENT, OR A MEMBER OF HIS FAMILY, OF THE IDENTITIES OF THE DOCTORS WHO WILL COLLABORATE IN THE CARE OF THE PATIENT.

Once a patient is referred for care and treatment, the surgeon is morally and legally responsible for his own preoperative diagnosis; for the decision to operate; for such preoperative care as has not been given; for the performance of the operation, and postoperative care as long as necessary. These responsibilities do not preclude consultation with colleagues. The patient should be referred back to his physician when he no longer requires surgical care.

The concept of surgical care as a team effort is important. The patient should be informed that he may be cared for by several individuals working under the direction of the responsible surgeon.

(b) SHOULD BE WILLING TO DISCUSS HIS FEE WITH THE PATIENT, OR A MEMBER OF THE FAMILY, PRIOR TO SUBMITTING A STATEMENT.

Discussion prior to submitting a statement may prevent misunderstanding and the resentment of patients about the surgeon's fee. Satisfactory agreement can usually be reached by a frank discussion of all considerations involved in the determination of a fee.

The fee of Fellows or Certificants should be commensurate with the services rendered and the reasonable ability of the patient to pay.

An exorbitant fee is one which exceeds either the value of the services rendered, or the reasonable ability of the patient to pay.

The value of the services performed depends upon the type of operation, the extent of the surgery and the special surgical skill required.

The surgeon should charge only for those services rendered personally by him or, under his direction, by his employees or surgical associates.

The referring physician, or consultant, should submit his individual bill for the services which he renders.

Reasonable ability to pay is the ability of the responsibile individual to pay without endangering the economic stability of the family.

To determine reasonable ability to pay, all expenses incident to the care of the patient should be considered. The economic level of the family should govern, regardless of the existence of insurance against the costs of medical care, except that an insurance benefit may be regarded as ability to pay that amount. Medical care insurance is not designed to serve as a platform upon which to erect an additional fee not justified by the economic level of the family.

When two doctors are to submit individual bills, and it becomes apparent that the total charges to the patient will exceed his reasonable ability to pay, it is proper that they confer and adjust their fees downward.

(c) PRESENT A STATEMENT FOR HIS OWN SERVICES TO THE PATIENT.

Payment should be made individually to each doctor regardless of whether separate bills or a combined itemized bill are presented. This principle applies with equal force to payment by a third party.

Ethically, a Fellow or a Certificant may not pay the referring physician for any services the latter performs in the care

of the patients, nor may a Fellow or a Certificant resort to a subterfuge to assist the referring physician to collect an unjustifiable fee, such as permitting the latter to render unnecessary services.

2. IN HIS RELATIONSHIP WITH PROFESSIONAL COLLEAGUES, a Fellow of The Royal College of Physicians and Surgeons of Canada, or a Certificant of the College should refuse to participate in, or countenance, any financial arrangement which would induce referral of a patient; refuse to permit a referring physician to collect his fee for him; refuse to collect the fees of other doctors collaborating in the care of the patient.

(a) REFUSE TO PARTICIPATE IN, OR COUNTENANCE, ANY FINANCIAL ARRANGEMENT WHICH WOULD INDUCE REFERRAL OF A PATIENT.

The responsibility of a physician to his patient requires that he place the interests of his patient above every other consideration. It should be understood that, in the referral of a patient for surgical care, the physician be guided only by the quality of care expected of the surgeon. Acceptance of any other inducement is a violation of the trust placed in him by the patient.

The patient must be protected from various forms of subterfuge which contribute to unethical inducement:

1. Division of the surgical fee between surgeon and referring physician. Division of a fee paid by an insurance carrier is as much fee-splitting as division of a direct payment by the patient.
2. Permitting the referring physician to collect the total bill from the patient and pay the surgeon.
3. Alternate billing of surgical patients, wherein the surgeon and referring physician collect and retain the entire fee from alternate patients.
4. Disproportionate reduction of the surgeon's fee to enable the referring physician to charge excessively for his services.
5. Payment of office rent on a percentage of professional income, particularly when the owners or lessees of the space can refer patients to the surgeon.
6. Lavish entertainment of referring physicians.

Acceptance of a rebate from a manufacturer or dealer, who has supplied a patient with a drug, appliance or other adjunct to treatment, is unethical. It is unethical for an ophthalmologist, or for an ophthalmic surgeon, to profit from the sale of glasses, or to profit from the services of an optician working either in his office or upon a referral basis.

It is also unethical for a Fellow or Certificant to accept a rebate from a clinical pathology laboratory, or a roentgenologist.

(b) REFUSAL TO PERMIT A REFERRING PHYSICIAN TO COLLECT THE SURGEON'S FEE FOR HIM.

The surgeon is not a subcontractor in the surgical care of the patient. Unless the surgeon presents his own bill to the patient, the latter has no way of knowing the amount of the surgeon's fee. Payment of the surgeon by the referring physician may invite fee-splitting and may result in an excessive bill to the patient.

(c) REFUSE TO COLLECT THE FEES OF OTHER PHYSICIANS COLLABORATING IN THE CARE OF THE PATIENT.

The patient is entitled to know how much he has been charged by each doctor with whom he has a contractual relationship. This can be assured only by each physician sending and collecting his own bill.

A surgical assistant, other than the referring physician, who stands in no other professional relationship with the patient, is an employee of the surgeon and can be paid an assistant's fee by the surgeon if this amount is indicated on the account sent to the patient.

In the submission of statements to patients, clinic groups and formal partnerships are regarded as single contractors, and may submit one bill for all services rendered by individual members. Accounts should be itemized so the patient will know the doctor rendering the service.

FORTHCOMING MEETINGS

UNIVERSITY OF TORONTO POSTGRADUATE COURSE IN OTO-LARYNGOLOGY

A graduate course in oto-laryngology will be presented by the Staff of the Department of Oto-laryngology on May 11, 12 and 13, 1961. They will be assisted by two distinguished guests, Dr. Philip E. Meltzer, Professor of Oto-laryngology, Harvard Medical School, and Chief of Oto-laryngology, Massachusetts Eye and Ear Infirmary, and Dr. W. G. Hemenway, Department of Oto-laryngology, University of Chicago.

The first session will begin in the afternoon of May 11, in the Royal York Hotel, Toronto, in association with the Section of Oto-laryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss, and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems

will be presented with special consideration of the new conceptions of responsibilities of this specialty in their management.

The fee for the course will be $40, and will include a complimentary dinner.

All enquiries should be addressed to the Director, Division of Postgraduate Medical Education, University of Toronto.

AMERICAN COLLEGE OF SURGEONS: FIFTH POSTGRADUATE COURSE ON FRACTURES AND OTHER TRAUMA

The Fifth Postgraduate Course on Fractures and Other Trauma, sponsored by the Chicago Committee on Trauma of the American College of Surgeons, will be held from April 19-22, 1961, at the John B. Murphy Memorial Auditorium, 50 East Erie Street, Chicago.

The course for 1961 is dedicated to Dr. Edwin Ryerson, an eminent orthopedic surgeon who was a leader in his field and for many years was actively associated with the Chicago Committee on Trauma of the American College of Surgeons.

The course will be presented by a distinguished faculty of 11 guest speakers and teachers prominent in the fields of trauma, from the five medical schools, and chiefs of services from leading Chicago hospitals who will discuss all phases of trauma; injuries to the head, face, chest, abdomen; genitourinary tract; plastic procedures on the extremities; skeletal traction versus adhesive traction; shock; the current prevention and treatment of gas gangrene; blood vessel injuries; major tendon disruptions; acute tendon injuries of the hand and forearm; ligamentous injuries to the knee; modern amputation techniques, and a new treatment for suppurative conditions secondary to trauma of the skeleton.

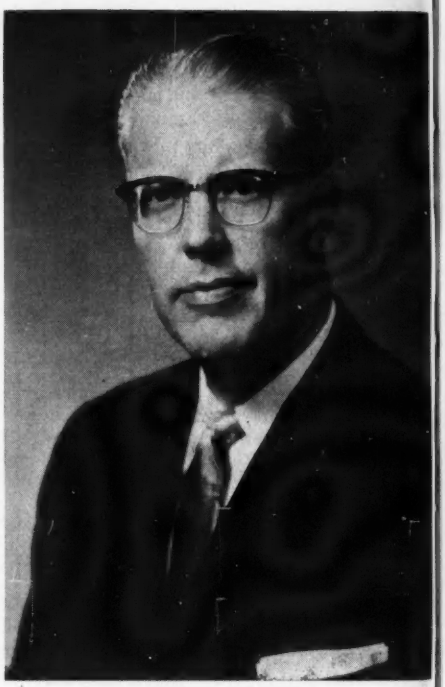
There will be panel discussions on athletic injuries, and fractures and dislocations of the tarsal bones. Many types of fractures and dislocations will be discussed. In addition, audio-visual programs, consultative periods, panel discussions, question-and-answer periods and eight presentations concerning various types of trauma will also form a prominent part of the program.

Distinguished visiting speakers will be Drs. William H. Bickel, Rochester, Minn.; Bruce J. Brewer, Milwaukee; James Barrett Brown, St. Louis; Edwin F. Cave, Boston; C. Howard Hatcher, Palo Alto; Charles C. Higgins, Cleveland; J. William Littler, New York; Owen E. Miller, Milwaukee; Austin T. Moore, Columbia, S.C.; Don H. O'Donoghue, Oklahoma City; and Robert A. Robinson, Baltimore.

The individual notices to physicians, which will be mailed well in advance of the meeting, will list suitable hotels in close proximity to the John B. Murphy Memorial Auditorium. The registration fee will be $75.00.

Dr. Sam W. Banks, Chairman of the Chicago Committee on Trauma, is Director of the course. Inquiries should be addressed to Dr. John J. Fahey, who is Chairman of the Committee on the Postgraduate Course on Fractures and Other Trauma, at 1791 W. Howard Street, Chicago 26, Illinois, U.S.A.

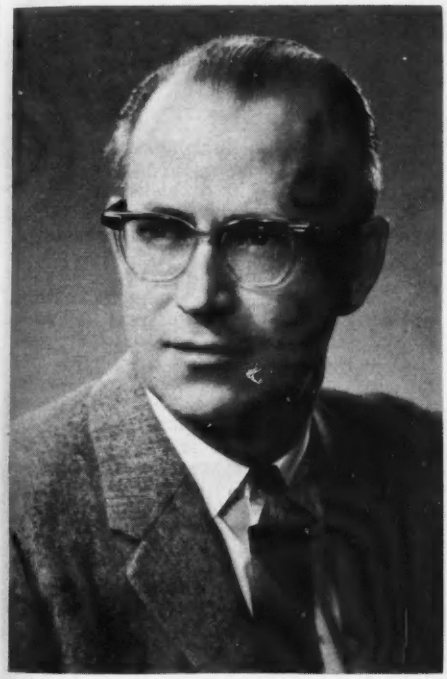
NOTICES



DR. WALTER C. MACKENZIE

With great regret we announce the resignation of Dr. Walter C. MacKenzie from the Editorial Board of the *Canadian Journal of Surgery*. Since the first number of the journal was published in October 1957, Dr. MacKenzie has contributed unstintingly of his efforts and his wisdom, a contribution that has been in no small way responsible for such success as the board

has achieved in the discharge of its functions. Dr. MacKenzie's ever-increasing duties and responsibilities as Dean of the Faculty of Medicine of the University of Alberta, required that he relinquish his post as Professor and Head of the Department of Surgery in 1960 and his resignation from the Editorial Board of the journal followed in consequence. The board records its sincere gratitude to Dr. MacKenzie for his contributions of the past and will sorely miss the benefit of his experience and sound judgment.



DR. ROBERT A. L. MACBETH

Succeeding Dean MacKenzie as Professor and Head of the Department of Surgery at the University of Alberta, and as that university's representative on the Editorial Board of the *Canadian Journal of Surgery* is Dr. Robert A. L. Macbeth of Edmonton. With the anticipation of a long and mutually gratifying association, the board welcomes Dr. Macbeth to membership in its ranks.

1961 MEDALLIST—THE ROYAL COLLEGE OF SURGEONS OF CANADA.



Royal Studio, Toronto

DR. JOHN STATON SPEAKMAN

The Annual Award in Surgery of the Royal College of Physicians and Surgeons of Canada was this year presented to Dr. John S. Speakman of Toronto in recognition of his studies on the structure of the trabecular meshwork and corneal endothelium in relation to the problem of resistance to outflow in open angle glaucoma. Dr. Speakman's investigations on this project were conducted in the Department of Ophthalmology of the University of Toronto and the Department of Pathology, Institute of Ophthalmology, London, England.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Calcium Metabolism and the Bone. Paul Fourman, M.D., D.Sc., F.R.C.P. Senior Lecturer in Medicine, Welsh National School of Medicine. 325 pp. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $10.25.

Clinical Orthopædics. Editor-in-Chief, Anthony F. DePalma, with the Assistance of the Associate

Editors, the Board of Advisory Editors, the Board of Corresponding Editors. No. 17. 387 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1960. $6.00 to sustaining members, $7.50 per individual copy.

Clinical Orthopædics Editor-in-Chief Anthony DePalma, with the Assistance of the Associate Editors, the Board of Advisory Editors, the Board of Corresponding Editors. No. 18, 293 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1960. $6.00 to sustaining members, $7.50 per individual copy.

Congenital Deformities. Gavin C. Gordon, M.B., F.R.C.S.E. Consultant Orthopædic Surgeon, Cumberland and North Westmorland; Orthopædic Surgeon and Adviser in Physical Medicine, Irton Hall School (N.S.S.), Cumberland, England. 128 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $6.35.

Klinische Chirurgie für die Praxis. In vier Banden. Band 1, Lieferung 4. (Clinical Practice of Surgery. In 4 volumes. Vol. 1, Part 4). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 366 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $12.85.

Modern Trends in Urology (Second Series). Edited by Sir Eric Riches, Past President British Association of Urological Surgeons (Home and Overseas), Surgeon and Urologist the Middlesex Hospital, London. 27 contributors. 287 pp. Illust. Butterworth & Co. Ltd., London, 1960. $14.00.

Nouvelle Pratique Chirurgicale Illustrée. Fascicule XVI. Directeur Jean Quénu. (New Surgical Practice Illustrated. Edited by Jean Quénu). 296 pp. Illust. G. Doin et Cie, Paris, 1960. 41NF.

Surgery of the Acute Abdomen. John A. Shepherd, *V.R.D.*, M.D., Ch.M.(St. And.), F.R.C.S.(Ed.), F.R.C.S.(Eng.), *Q.H.S.* Consultant General Surgeon, Liverpool Region. Formerly Lecturer in Surgery, University of Liverpool. Surgeon Captain R.N.R. Foreword by Sir Zachary Cope, M.D., M.S., F.R.C.S. 1228 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, 1960. $17.00.

The Surgical Treatment of Portal Hypertension, Bleeding Esophageal Varices and Ascites. M. Judson Mackby, M.D., D.A.B.S., F.I.C.S. Staff Surgeon, Kaiser Foundation Hospital, San Francisco, California, and Staff Surgeon, Permanent Medical Group. Foreword by Emile Holman, M.D., Professor Emeritus of Surgery, Stanford University Medical School. American Lecture Series No. 388. 250 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $11.50.

Transactions of the International Society of Plastic Surgeons. Second Congress, London, 1959. Edited by A. B. Wallace, M.Sc., F.R.C.S.(Ed.) Translations of abstracts into French by Michael N. Tempest, Ch.M., F.R.C.S.(Ed.), German by W. Grossman, M.D., and Spanish by R. P. G. Sandon, F.R.C.S. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto. 1960. $15.25.

The Urological Aspects of Bilhariasis in Rhodesia. R. M. Honey, M.B., F.R.C.S.(Ed.), Consulting Urologist, Salisbury Hospitals Group, and M. Gelfand, C.B.E., M.D., F.R.C.P., Consulting Physician, European and African Hospitals, Salisbury, Rhodesia. Foreword by D. M. Blair, O.B.E., M.D., D.P.H., Secretary for Health, Federation of Rhodesia and Nyasaland. 71 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1960. $1.30.

BOOK REVIEWS

(See also pages 276, 292, 320, 323, 337 and 382)

HYPOTHERMIA FOR THE NEUROSURGICAL PATIENT. Antonio Boba, M.D., Associate Professor of Anesthesiology, the Albany Medical College of Union University, Albany, N.Y. 124 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $6.50.

This concise, well written monograph should be of particular value to neurosurgeons and anesthetists.

The general physiological responses to hypothermia are described and illustrated with great clarity and authority. All important references are included.

Various methods of achieving hypothermia are briefly outlined. Mechanisms of heat transfer and temperature gradients which result from the various methods are well described and illustrated. The detection and management of the problem of "shivering" is well handled. The effect of hypothermia upon the central nervous system is well covered including problems of volume, pressure and ability to withstand injury.

The various parameters which the anesthetist must observe and manage in clinical hypothermia, are well described.

The hazards and complications are frankly outlined.

This book should be of great interest to all who have any concern with experimental or clinical hypothermia.

LES ENTRETIENS DE BICHAT 1960. Chirurgie-spécialités. (Colloquium of Bichat, Surgery and Specialities, 1960). Edited by R. Gueulette, J. Hepp, Y.-J. Longuet and M. Roux, chirurgiers de l'Hôpital Bichat. 557 pp. Expansion Scientifique Française, 1960.

Ce volume est un recueil de nombreux articles par des auteurs différents, couvrant plusieurs sujets de chirurgie générale et des spécialités chirurgicales. Bien qu'il soit impossible de donner une opinion d'ensemble sur un tel ouvrage, on peut affirmer que la plupart des travaux résument de façon concise un problème donné.

L'ouvrage comprend deux parties: chirurgie générale et spécialités. Mais, on peut reprocher aux éditeurs de n'avoir pas groupé à l'intérieur de cette division les articles concernant un même sujet. Une table analytique, à la fin de l'ouvrage, corrige en partie cette lacune.

En chirurgie vasculaire, deux articles traitent des pontages artériels. Mentha démontre l'utilité de la prise de la tension artérielle per-opératoire dans les différents segments des artères intéressées; tandis que Natali insiste sur le genre d'activité du malade quand il pose les indications d'une prothèse artérielle. Léger et Mouktar ont publié un bon résumé de la question de l'anévrysme de l'artère splénique. D'autres articles traitent de l'hypertension portale, des complications thrombo-emboliques et du syndrome post-phlébitique.

Plusieurs problèmes sont discutés en chirurgie de l'appareil digestif tels que les adénites mésentériques, l'ulcus duodénal, la lithiase cholédocienne, le syndrome de Zollinger Ellison . . . Bernard Duhamel présente les possibilités de sa technique consistant en une exclusion du rectum avec abaissement rétro-rectal et trans-anal du côlon.

En conclusion, l'ouvrage est une collection de travaux d'un groupe imposant de chirurgiens et une aide à qui veut les solutions de ces auteurs aux problèmes qui y sont traités.

CHEMICAL OSTEOSYNTHESIS IN ORTHOPÆDIC SURGERY. Michael P. Mandarino, M.D., Associate, Department of Orthopædic Surgery, Hahnemann Medical College and Hospital. 72 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $5.00.

In this small book the author presents an enthusiastic brief for the use of bone glue. The glue under discussion is a polyurethane foam used under the W. S. Merrell & Company trade name of Ostamer. This substance has the necessary properties. It is strong, non-toxic, adheres to bone, expands out into irregular spaces, provides a lattice into which bone many grow and is relatively easy to use.

The author outlines rather extensive indications for the use of Ostamer. The prime use seems to be in non-union of bone in which previous operative measures have failed. Other indications considered are pathological fractures, bone cysts, acute fracture prone to complications, spinal fusions and other joint fusions. The technique of the use of Ostamer is simple. A dry field is necessary to allow strong bonding of glue to bone. With this in mind, one wonders how good fixation can be achieved in spinal fusions. If the medullary canal of the long bone is small, the author has used intramedullary rods for additional support. Emphasis is placed on the use of a large mass of Ostamer.

To support his claim for Ostamer, the author outlines his experience with 50 personally treated patients. The results are impressive. Many patients had non-union of long bone fractures previously treated unsuccessfully by operation. The first tibial fracture in this series was operated upon in 1956. All patients were allowed early ambulation. Of the 50 cases, all but three had successful results. It is pointed out that even in the presence of infection Ostamer may be used. It is apparent that the author is an enthusiast, and his own recorded results with the use of Ostamer are very impressive.

With general use it is likely that bone glue will find an important but more restricted use in the treatment of difficult non-unions and pathological fractures of long bones.

NOUVELLE PRATIQUE CHIRURGICALE ILLUSTREE. Fascicule XV (New Surgical Practice, Illustrated, Fascicle XV). Edited by Jean Quénu. 286 pp. Illust. G. Doin et Cie, Paris, 1960. 39NF.

Cet ouvrage est un classique et est très connu des chirurgiens de langue française. Il se compose de plusieurs fascicules, chacun décrivant sous forme d'atlas quelques techniques opératoires. A certaines occasions, l'histoire d'un cas et quelques commentaires sur le traitement de la maladie sont notés.

Le fascicule XV décrit l'ablation d'un neurinome intrathoracique, la myoplastie pour incontinence anale, la kysto-jéjunostomie pour pseudo-kyste du pancréas, la myomectomie abdominale, la côlo-cystoplastie et finalement l'aponévrectomie palmaire.

A propos de la myoplastie à l'aide du muscle droit interne pour corriger une incontinence anale, Dubost note que les résultats se détériorent avec le temps et qu'il ne faut pas se fier à des statistiques faisant état de succès trop précoces.

De plus, Perrotin conseille la dérivation interne par kysto-jéjunostomie en Y quand le pseudo-kyste du pancréas n'est pas réséquable.

En résumé, ces manuels de technique chirurgicale sont bien faits en ce qui concerne la description des différents temps opératoires. Cependant, on peut reprocher aux éditeurs de n'avoir pas groupé les différentes techniques selon les régions anatomiques, mais de les avoir disposées, semble-t-il, au hasard.

MEMORY, LEARNING AND LANGUAGE. The Physical Basis of Mind. Edited by William Feindel. University of Saskatchewan Jubilee Symposium, 1959. 69 pp. Illust. University of Toronto Press, 1960. $2.00.

This book consists of six essays comprising a symposium that was designed to acquaint intelligent laymen with some views of the function of the brain that have an important bearing on the processes of education. Sur-

geons with an enquiring mind, who are interested in the greater potentialities of the human brain, and whose normal reading does not include recent neurological literature, should find this small book of interest and a stimulus for further enquiry. The essays were given originally in 1959 as lectures at the 50th anniversary celebration of the University of Saskatchewan. The humanistic, anatomical, chemical, cybernetic, mechanical, and physiological viewpoints of mental activity are each outlined in turn. The heritage from Ancient Greece and Rome in our modern concept of memory is discussed by Dr. J. F. Leddy. Dr. William Feindel, the editor, sketches the microscopic anatomy of the cerebrum, the significance of electroencephalography and some mechanistic views of cerebral activity. Dr. A. Hoffer contributes a short discussion of the strange effects on mental activity of hallucinogenic drugs. He includes rather an uncritical reference to the place of these drugs in the treatment of alcoholics. Dr. J. W. T. Spinks, President of the University of Saskatchewan, describes self-regulating machines, with a fascinating example of a self-propelled machine which is directed on its course by a rotating Geiger tube and is designed to follow the perambulations of a burrowing worm that is tagged with radioactive cobalt. Dr. Arthur Porter describes the arithmetical and logical operations of automatic computers and control systems. In the concluding essay Dr. Wilder Penfield outlines the physiological process of learning to speak and his well-known arguments in favour of teaching multiple languages in early childhood.

THE TRANSPLANTATION OF TISSUES AND ORGANS. Michael F. A. Woodruff, M.D., M.S.(Melb.), F.R.C.S., F.R.C.S.E., F.R.A.C.S. Professor of Surgical Science in the University of Edinburgh. 777 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $28.00.

A distinguished professor of surgical science has in this book produced a most comprehensive single volume covering the widest phases of the exciting field of tissue and organ transplantation. In a rapidly advancing field, encompassing basic and clinical science, Professor Woodruff has met the difficult problem of organization and classification of material effectively. He has resisted the temptation to fantasize in a realm where imagination and ingenuity have so frequently played a role.

There is little doubt that the next decade or two will bring transplantation into all fields of medicine and this text promises to become a classic. Of the 110 pages of references, many are timely and for the most part well chosen. Current concepts of basic principles and techniques are presented clearly enough for even the casual reader to understand. In keeping with easy readability, long words and sentences are rarely used, where short ones will suffice.

Terminology is defined early and the material flows smoothly thereafter. Homotransplant survival, homograft immunity, experimental procedures and heterotransplant experiences are lucidly presented. Specific information regarding techniques of obtaining homotransplant materials now in use (artery, cornea, bone and so on) is well presented. The chapters on transplant of hematopoietic tissue, endocrine tissues and kidney are outstanding.

The author chose wisely not to dwell on blood transfusion (certainly a tissue transplant) since "there are already numerous books on the subject", and one wonders whether the chapters relating to plastic surgery, reconstructions of the alimentary tract and ureterointestinal anastomoses are, though well done, in keeping with the spirit of the book.

This classic is heartily recommended for the library of those working with tissue transplantation but will deservedly enjoy vigorous general circulation from the medical library shelves.

DAS SYSTEM DES DUCTUS THORACICUS UND DIE ERKRANKUNGEN DER REGIONALEN GEFASSE. Eduard Alther, with a preface by Prof. Dr. R. Nissen, Basel, Switzerland. 200 pp. Illust. Benno Schwabe & Company, Basel and Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $9.00.

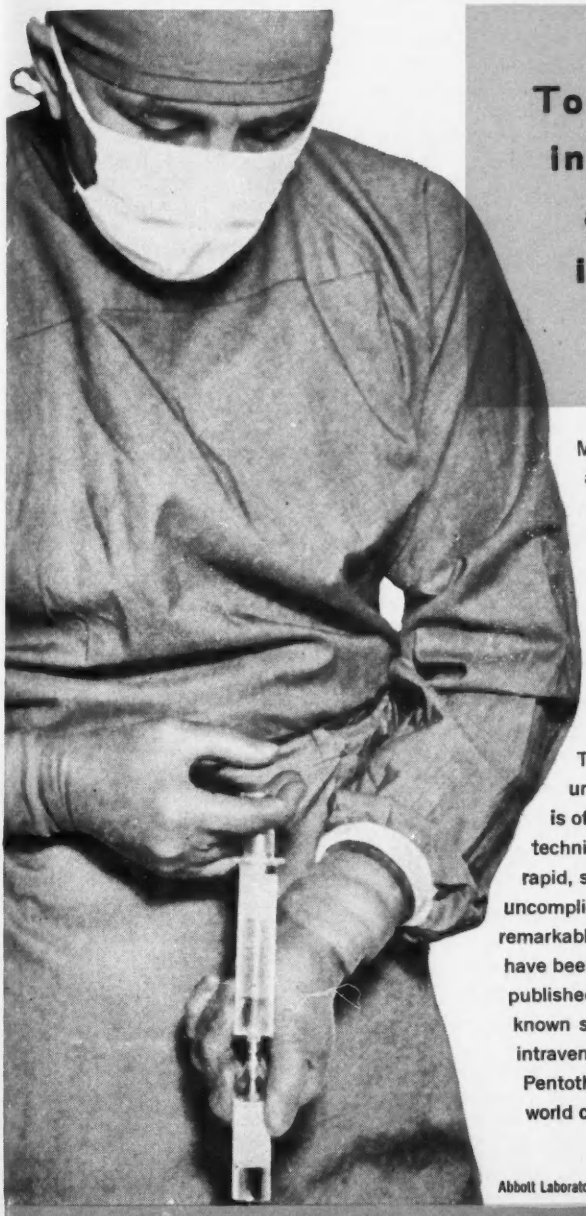
Studies of the lymphatics have been constantly bypassed in medical research projects in the past. It is therefore not surprising to notice that medical men are puzzled when confronted with problems involving this system.

The surgeon, noticing an accumulation of chylous fluid, frequently faces a situation for which he has no solution at hand. To fill this gap in medical knowledge, the author has undertaken the exploration of the thoracic duct system.

The importance of this work becomes apparent by the fact that this monograph has received the support of the Swiss National Fund for Scientific Research. As underlined by Professor Dr. Nissen of Basel in his preface to this book, the reasons for the accumulation of chylous fluid, apart from traumatic causes, remain in large part unsolved. Existing knowledge about the lymphatic system was mainly concerned with its relationship to the defense mechanisms of the body against infections.

Therefore, the author's explanations of the hitherto unknown connections between the normally functioning lymphatics and their pathological changes are of value. In order to facilitate this understanding, the author develops original ideas about the embryonal development of the lymphatic system.

(Continued on page 400)



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(Continued from page 398)

Of interest to the reader will be the numerous excellent histological illustrations incorporated in this book. By analyzing the changes brought about by disease in the lymphatic vessel-tissue relationships, Dr. Alther contributes to a more comprehensive understanding of the lymphatics and their functions.

From this point the author proceeds to describe the different forms of treatment necessitated by the interruption of the circulation of chlye. In addition, this book, published in the German language, cites detailed case histories with therapeutic measures to be taken in order to provide the reader with a comprehensive understanding of the lymphatics and the problems in this field which may be encountered in medical practice.

KLINISCHE CHIRURGIE FUR DIE PRAXIS. In vier Banden. Band I. Lieferung III. (Clinical Practice of Surgery. In four volumes. Vol. 1 Part III). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 618 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $12.85.

This edition deals with the diseases and surgical treatment of blood vessels, lymphatics and endocrine glands. Surgery as taught to medical students in continental Europe consists mainly of a mixture of pathology and medicine with additional lectures on fractures.

This book, intended to prepare European students for their surgery examination and to help the general practitioner in the daily problems confronting him, keeps within those limitations. Surgical problems, once diagnosis is established, come within the province of the specialist. Therefore, text books on surgery published in Germany cannot be compared with Canadian editions. Their purposes are different.

Consequently one will find good coverage of descriptive pathology, laboratory techniques and differential diagnosis in the European works, as achieved by the authors of this book.

It is to be regretted that B. Löhr, author of the chapter on blood vessels, in his endeavour for perfection to establish diagnosis, loses himself in subdivisions and statistics. In order to demonstrate Raynaud's disease, for example, one colour picture would have done much more than the lengthy and confusing tabulation. On the other hand, practically no detailed treatment is given for varicose veins. Their surgical treatment is mentioned and the reader is directed to look up that subject in the books of Sigg, Schörcher, and others.

The same applies to the equally important office procedure for varicose ulcers, for which few suggestions for treatment are offered in

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Edited by SIR ERIC RICHES, M.C.M.S., F.R.C.S. Past President, British Association of Urological Surgeons. **Price $14.00**

This Second Series of MODERN TRENDS IN UROLOGY spans the intervening years since the first (1953) progress volume. Urology is a progressive science and in these years great advances in both diagnosis and treatment have been made reflecting the close collaboration which exists between surgeon, physician and laboratory worker. The advances in pathology and biochemistry and a knowledge of the influence of hormones and of nuclear sexing are all subjects with which the modern urologist must be familiar; he must seek the aid of an experimental surgeon in the search for the solution of the immunological problems which limit the full development of renal transplantation.

The contributions strike a realistic balance between medicine and surgery making the volume not only of great interest to urologists, but also of much value to biochemists, radiologists, radiotherapists, gynaecologists, pathologists, general surgeons and physicians.

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this book. One also misses descriptions of the surgical treatment of aneurysms of the aorta.

The chapters on the endocrine glands are good, dealing mainly with symptomatology and medical treatment. They are comprehensive and record the latest developments in endocrinology in a manner which is easy to stand.

Although published in 1960, there is no mention of oral hypoglycemic agents in the treatment of diabetes mellitus.

On the whole this book is to be recommended to the general practitioner in continental Europe.

KLINISCHE CHIRURGIE FUR DIE PRAXIS. In vier Banden. Band III, Lieferung 2. (Clinical Practice of Surgery. In four volumes. Vol. III, Part 2). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 360 pp. Illust. George Thieme Verlag, Stuttgart, W. Germany. Intercontinental Medical Book Corporation, New York, 1960. $9.50.

This book deals with the stomach and duodenum, their anatomy, histology, vascular and nervous supplies, physiology, pathophysiology, diseases and surgery.

It seems, therefore, that the authors of this volume have advanced the art of editing surgical textbooks by trying to convey to readers under one cover as complete a picture as possible of the organ concerned. Furthermore, this book, originating from West Germany, stimulates interesting comparisons with daily medical practice in Canada.

An impressive number of British and North American authors are mentioned in this book. It appears that western Germany has adopted numerous medical items from the western hemisphere in the postwar years. For the sake of perfecting future editions, various shortcomings might well be pointed out here.

The frequent interpolation of bibliographical data into the text — nearly one to every line—interferes with the even flow of reading. An appendix would compensate for their omissions from the text.

In the chapter on peptic ulcer the authors have failed to enumerate proper mastication of food and dental care as important factors for the prevention and treatment of ulcers. Also, abuse of tobacco is only mentioned in passing. In the reviewer's opinion estrogen therapy for chronic peptic ulcer merits inclusion in the text.

The different indications for gastrectromy are crowded into a few lines and, to make things worse, references to other pages of the book are used. A better arrangement and spacing of the text would help the reader to retain this important matter in his mind. Finally, Virchow's gland should be mentioned among the findings leading to the diagnosis of cancer of the stomach.

(Continued on page 403)

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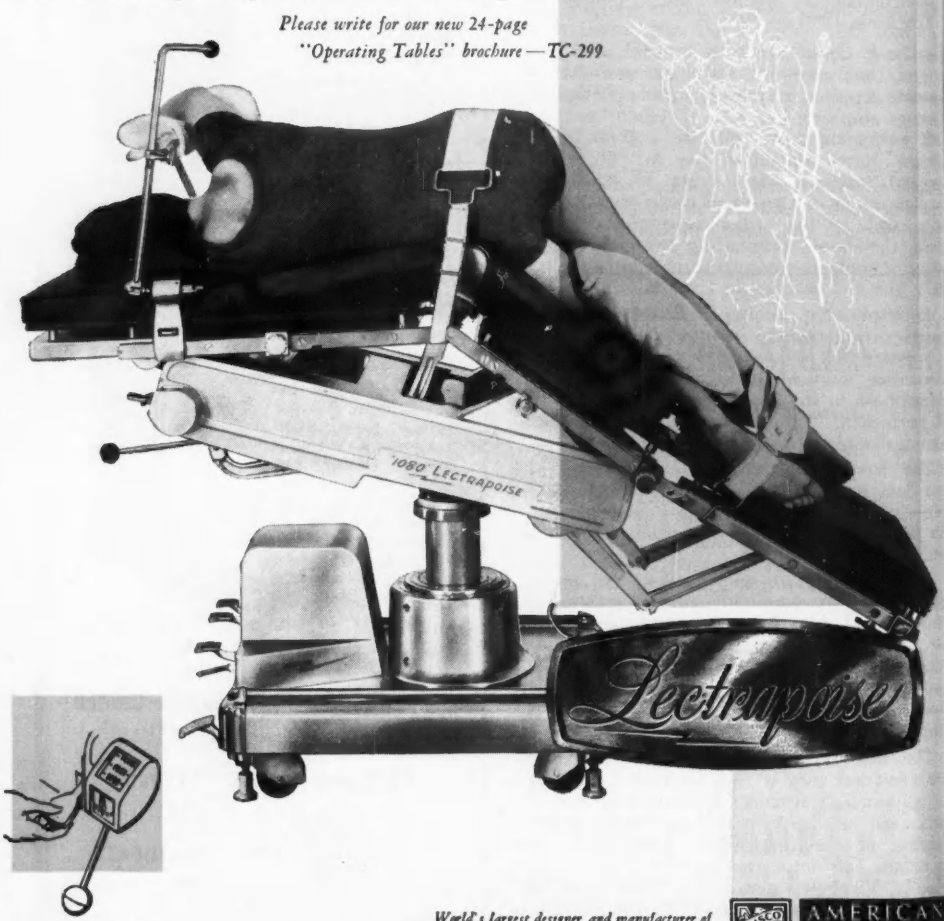
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(Continued from page 401)

Apart from these few corrections this book offers the advantage of complete coverage of the subjects concerned in accord with the latest developments achieved in the medical world.

The chapters on "dumping syndrome", as well as the historical survey, listing milestones in gastrointestinal research over the last 300 years, deserve special praise and conclude this remarkable book.

PRINCIPLES OF ORTHOPÆDIC SURGERY. Paul C. Colonna, M.D., Emeritus Professor of Orthopædic Surgery, University of Pennsylvania Medical School. 799 pp. Illust. Revised edition. Little, Brown & Company, Boston.; J. P. Lippincott Company, Montreal, 1960. $22.00.

In this complete revision of his earlier work, Dr. Colonna has briefly covered the entire field of orthopedic surgery in adults and children on a regional basis. The opening chapters contain a summary of the physiology of bones and joints, a method of musculoskeletal examination and a section on general orthopedic pathology. Each region of the body is then discussed in a separate chapter pointing out the pertinent anatomy and special examination before discussing diseases and general anomalies, injuries to soft tissues and fractures as they apply to each particular region. The final chapters of the book deal with neuromuscular disorders, bone tumours and orthopedic appliances.

It is well indexed and although there are few if any references to works published within the last four or five years, most of the classic references have been included, making it a useful springboard for further background reading on any desired subject.

While any volume of this scope will contain sections with which the reader may disagree such as those on the surgery of cerebral palsy and tuberculosis, a very reasonable approach to the subject has been presented, obviously based on years of experience.

It should prove particularly valuable for residents in orthopedic training programs and will be a useful addition to the library of the general surgeon dealing with bone or joint problems as well as to the orthopedic surgeon.

ILLUSTRATING MEDICINE AND SURGERY. Margaret C. McLarty. 158 pp. Illust. E. & S. Livingstone Ltd., Edinburgh; The Macmillan Company of Canada Limited, Toronto, 1960. $6.35.

The text of this book, capably expressed and beautifully illustrated, is composed of well-known precepts and principles relative to medical illustration. It cannot be said to offer anything new. To the scientist or doctor with a flair for drawing it provides technical hints, suggests equipment and materials. The technical hints are neither simple nor full enough for the uninitiated and being scattered here and there are not easy to pin-point. The equipment and supplies suggested are well presented with names of dealers and prices but these are useful mainly to those living in the British Isles, many of them being obsolete by Canadian standards and not procurable on this side of the Atlantic. The value of the book lies in the comprehensive picture it gives of medical illustration, past and present. To art and science students at the undergraduate level and vocational guidance directors, it can be highly recommended.

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*Thomson, G.: The two aspects of Science, *Science*, October 14, 1960.



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HISTORY OF CANADIAN SURGERY

ABRAHAM GROVES OF FERGUS:

The First Elective Appendectomy?

CHARLES W. HARRIS, M.B., F.R.C.S.,* *Toronto*

THE FIRST operation for removal of the vermiform appendix because of previously diagnosed disease of that organ was performed by Abraham Groves in a farmhouse near Fergus, Ontario, on May 10, 1883. The "barbarous term appendicitis" had not yet been coined. Groves and his contemporaries thought that this was the first occasion on which this operation had been done in Canada. It may have been the first in a far wider sphere. This is not his only claim to fame. He had other "firsts", but even without them, his strenuous unselfish life of practice in the Ontario countryside is worthy of recognition.

His family was Irish.[1] Abraham Groves, the father, after whom the doctor was named, was a lad of 17, when on May 21, 1825, the brig *John Barr* sailed from Cork, Ireland, for Quebec. With him as head of the family was Thomas, aged 20, a "reduced farmer" according to the ship's passenger manifest, two younger brothers and their mother, Margaret Groves, aged 40. They and some 2000 others had been recruited from County Wicklow by the British Government for settlement in Upper Canada and their ship was one of many that sailed at that time. According to the records the ships made a fast passage to Quebec losing only 15 passengers en route. This was in sharp contrast to the high mortality among the Irish immigrants a decade or so later when cholera was rife. The migration was under the direction of Peter Robinson, brother of Sir John Beverley Robinson, Chief Justice of Upper Canada. With government paternalism they continued their journey to Cobourg, then across the land to the mouth of the Otonabee River, where a week was spent constructing a boat to continue to the uninhabited Scott's Plains, later to be named Peterborough after Peter Robinson. This paternalism continued with supplies

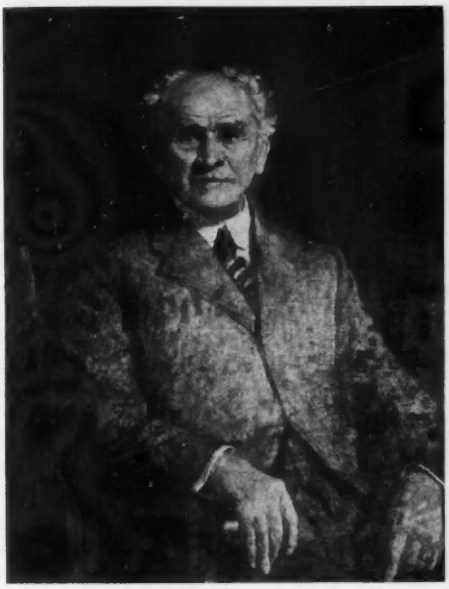


Fig. 1.—Dr. Abraham Groves, from a portrait by Sir Wylie Greer.

of food and tools. It paid off a decade later when these same loyal Irishmen marched in a battalion to Toronto at the time of the rebellion.[2] Thomas Groves, as head of the family, received his 200 acres in Emily township where his name may be found on the assessment rolls. Abraham Groves senior married a daughter of a veteran of Lundy's Lane.[3] Dr. Abraham Groves was born on September 8, 1847, the fifth and eldest surviving child of this marriage.

When he was five years old, Abraham's father took land on the Garafraxa Road, a few miles from Fergus on the edge of the recently settled Queen's Bush which reached thence to Georgian Bay and Lake Huron. According to evidence given to the British Parliamentary Committee on Emigration, the Irish succeeded best when they were intermingled with English and Scottish settlers. The Groves thrived among the

*Suite 342, Toronto Western Hospital, Toronto 2B.

Scots of Fergus. The life span of the younger Abraham Groves covered the period when the countryside and towns grew from infancy to maturity.

He attended the public and high schools at Fergus and then went on to the Toronto School of Medicine, from which he graduated in 1871. During his early years in Toronto he was a friend of William Osler, an attraction perhaps of opposites; Groves from the farm and Osler with the cultural background of an Anglican parsonage. Their futures were to be antipodean, Osler to high places and Groves back to the countryside to sharpen his wits alone. They had in common the abilities to work, to write and to be curious. They shared and paid tribute to the same teacher, James Bovell, one-time pupil of Astley Cooper, a man of rich mental endowments and catholic interests whose humanitarianism was to lead him from medicine to the priesthood. Bovell lectured in physiology and pathology in the medical school and at the same time retained the chair of natural theology in Trinity College. It was the clinical shortcomings in Toronto that took Osler to McGill,[4] but Toronto has never lacked a good department and good teachers in anatomy. J. H. Richardson was the professor of anatomy from 1850 to 1902. In fact the anatomical laboratory was the only laboratory in the school. It is a fair presumption that Groves made good use of it. What better preparation for a pioneer surgeon! Groves also paid tribute to Dr. W. T. Aiken, his lecturer in surgery, who was practical in everything. "His teaching made surgery educated common sense". During his undergraduate days Groves spent one summer as assistant to a Toronto doctor. This was the extent of his preceptorship.

He returned to Fergus to practise in April 1871, six months ahead of the first passenger train to Elora and on to Fergus.[5] There, uninhibited by the shadows of authority, he had plenty of scope for his able, daring and self-reliant qualities. Birth on a frontier had its advantages. Sixty-four years later he wrote a book "All in the Day's Work".[6] This is autobiographical and delightful reading, but leaves one with the feeling that perhaps these stories have gained in the telling. Fortunately, Groves from the start of his practice was a frequent contributor to medical journals and his statements can be checked with his contemporary printed records. They are found to agree.

It would be a brash young man, who, within weeks of the commencement of his practice, would be giving a bled-out woman a blood transfusion, using a rubber syringe. The fates were kind; it succeeded. Three years later, daring the raised eyebrows of his colleagues and public hostility in case of failure, and never having seen an abdomen opened before, he did his first ovariotomy. Nor did he leave his ligatures long and fix the pedicle in the wound, but drain it he did. Soon after he was arguing the futility of drainage. Groves' first four published papers were on the subject of ovariotomy. It is surprising the hold that this operation had on surgeons' minds from the time of McDowell. It became Groves' habit, his fetish almost, after every laparotomy to flush the abdomen with gallons of boiled water. His reason was that typhoid fever and other infections were carried by water. Evolution did not begin with Darwin or infection with Pasteur. Lister's teaching had not then been generally accepted. Groves was naturally a clean man; he always remained a meticulous dresser. He was like the unusually successful Yorkshire veterinary of a previous century, who when he arrived at a farmhouse sent everybody out and remained alone in the kitchen for some mystic rite. On his death-bed he was asked what he did when alone and he confessed, "I boils me tools". Groves laid great stress on the preparation of his hands and the operating-room environment.

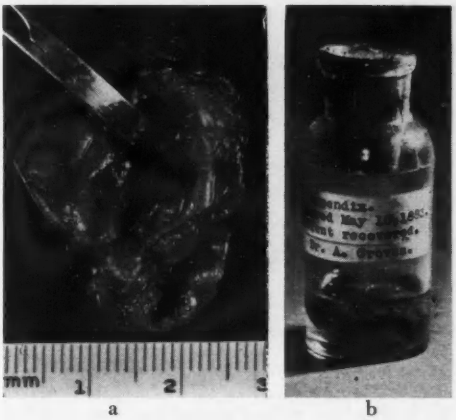
Groves' early surgery was done in farmhouses, most of them of a primitive type. It was the second stage of development of the Ontario farmhouse, the "good and sufficient" log cabins had yielded to frame houses as a result of the increase of the price of wheat during the Crimean War. Some of them boasted brick veneers. The only convenience was the water supply from a stone well. A kitchen table or boards placed on trestles became the operating table, milk-pans were the substitutes

for basins, and the kettle was the source of sterile water. Whenever possible preparations would be made beforehand, unneeded furniture pushed aside and covered with sheets, the table moved into the cleared room and the water boiled. The usual surgery was the surgery of urgency; operations for perforated ulcers, ruptured ectopic pregnancies, occasionally a Cesarean section or operations for rupture of the spleen or kidney. Groves described patients with depressed fractures of the skull requiring trepanning. He also spoke of laminectomies, and on occasion he operated on a mastoid or removed a cataract. From his experience he contributed papers on renal colic and prostatectomy. On April 20, 1878, in a bedroom in the old Bullfrog Tavern in Guelph, he operated on a 300 lb. man and removed six large calculi from the bladder by the suprapubic route. So far as he knew, it was the first time the suprapubic route had been used in Canada.

Groves maintained that in most operations few instruments were needed. To emphasize this point he once removed an appendix using only a Hagedorn needle. When he needed an otherwise unobtainable instrument, a bone plate for instance, it would be made for him by the local blacksmith. When he operated on a compound fracture, he always approached the fracture through a fresh wound. Many have paid tribute to the wizardry of his fingers. Having made the incision, the scalpel was laid aside and his delicate fingers completed the dissection.

Like all country doctors Groves was a good judge of horses, which he loved. At first he covered his practice of 50 miles' radius on horseback with saddlebags, later by buggy or cutter and ultimately by motor car. He finally controlled his travelling by founding a hospital, which soon instituted its own training school for nurses. This was the Royal Alexandra Hospital, later given to the town of Fergus and now known as the Abraham Groves Memorial and Community Hospital.

Dr. C. H. Ewing,[7] of Detroit, while an undergraduate, spent several summers with Dr. Groves. He tells of one particularly memorable day—



a b

Fig. 2.—The appendix removed by Abraham Groves, May 10, 1883, together with the labelled container in which it was stored until 1961.

"At six o'clock in the morning we left home in a Model T with four or five sets of instruments. At Dundalk we did an appendectomy, then on to Mount Forest where a leg was amputated for sarcoma; to Drayton for an appendix. There I was requisitioned for the anesthetic. It was my first, but Groves kept careful watch. One nod meant "Give more ether", two nods meant "Stop". When we got home, Mrs. Groves said, "I hope you fed that boy". I went to bed. Dr. Groves went to the hospital to do another job."

To the historian, the most interesting feature of Groves' life is his first planned removal of the appendix. Although appendicitis has always been with us it was only in the 1880's that it was universally recognized as the common cause of abscesses in the right lower quadrant, and "typhlitis" and "para-typhlitis" were sent into limbo. About 1820 two Frenchmen had described the disease, recognizing the cause and suggesting the cure. Their temerity had been borne out by the authority of the formidable Dupuytren. In opening an abscess Groves had once seen a gangrenous appendix lying free in it. But let Groves tell his own story—

". . . On May 10, 1883, I was called to see a boy, about 12 years old, suffering from pain and tenderness in the right iliac region. After examination I advised that an operation should be done immediately,



Fig. 3.—Low power photomicrograph of the Groves' appendix prepared in January 1961. (*Courtesy of Professor John Hamilton*).

to which the father consented. On making an opening an inflamed appendix was found. This was removed by first ligating the organ at its origin and also the appendiceal mesentery, which was then cut through. The appendiceal stump was sterilized by means of a probe heated in the flame of a lamp, and after thoroughly irrigating the abdominal cavity, the opening was closed in the usual manner, without drainage.

"On the third day, when I went to see the boy, he was doing well, but his father was very much dissatisfied. A neighbour, suffering apparently from the same disease, had been treated by poulticing and had recovered. The father told me that if I had known how to treat the case properly no operation would have been necessary, and that if the boy did not recover, it would not be well for me. Fortunately the patient recovered.

"At a meeting of medical men a short time later, I referred to the above operation and to this method of treating such cases, but found no supporters. In fact, one doctor went so far as to say that if such treatment became widespread the death rate would be appalling. In spite of this I continued the treatment successfully. So far as I am aware this was the first time that an appendix had ever been removed."

Henry Hancock,[8] surgeon to Charing Cross Hospital, in 1848 performed the first recorded successful operation for peritonitis due to disease of the appendix. Richard Hall[9] was the surgeon who first reported a case of survival after the removal of a perforated appendix. T. G. Morton has been credited with being the first to operate deliberately and remove the inflamed appendix after correct diagnosis in April 1887.[10] Sir Frederick Treves is said to have performed the first in Great Britain in 1888. Abraham Groves performed his operation for this disease four years ahead of Morton.

Dr. Groves' colleagues, whose lifetimes have overlapped ours, have always stated that Groves' removal of the appendix was the first in Canada. In 1922, in a paper in the *Canadian Medical Association Journal*, entitled, "Evolution of surgery as I have seen it in my own practice", he refers to his operation in 1883. This quotation was taken from his autobiography, "All in the Day's Work",[6] published in 1934. As noted, all his statements in that book have been verified by his contemporary publications. Groves was a man of undoubted probity and, as always, this is the greatest argument. He kept the specimen of this first appendix suitably preserved in some vinous spirit in a bottle which remained among his possessions. Mrs. Groves has donated this specimen to the museum of the Toronto Academy of Medicine.

Following the publication of his book, Dr. Groves received letters of commendation from the surgical great of the world, among them Lord Moynihan, Sir Alfred Webb-Johnston, Sir John Fraser, W. J. Mayo, George Crile, and Harvey Cushing. In their reviews of his book, both the *Lancet* and the *British Medical Journal* expressed concurrence in his work. No record of his contemporary publication of a report on this first appendectomy has been found. This is surprising because of his habit of writing. It is possible that he submitted a report and the editor considered it nonsense. Ephraim McDowell, who also lived on a frontier and with whom many comparisons might be made, did not publish reports of his successful ovariotomies until seven years had passed.[11] McDowell made only two contributions to medical literature, Groves made many. But even without a Groves or a McDowell, laparotomies were to become commonplace.

Those of his period claimed that Groves was ahead of his time. This again is substantiated by his writings. His unembel-

lished style was clear and direct, his opinions did not vacillate. His earlier reports were pleas against a negative attitude towards those conditions that might be cured by positive action. His article on the radical cure of hernia, published in 1903, had a modern sound. In it he stressed the importance of the fascial layers in the causation and the cure of this condition. As he grew older he philosophized more. Many now would agree with him on the influence of tobacco in the production of certain diseases; fewer would care to agree with him on the lack of any benefits from alcohol, but at least his personal life was consistent with his views. Few would agree with him on his rejection of hospital standardization by a "foreign authority" or in his opinion that heels on shoes were an evil.

The *Canada Lancet* was the early vehicle for his publications. Of all Canadian medical periodicals from 1870 to 1890, this is now the most interesting to read. Its contributors were from the towns of Ontario which then boasted a more advanced local culture than is to be found in this more mechanical age. Its editor plagiarized freely. One wonders if Lawson Tait knew the frequency of his contributions. Editorially, the *Canada Lancet* did not exclude meaningful language as it took on the able and cantankerous George Brown's *Globe* in its opposition to the new medical act. Then, as now, politicians knew where the votes were.

After 60 years of practice and service to his community, and with his eyesight failing, Groves retired. The little man with the brown curly hair and brown eyes, who never walked but always ran, finally sat for a portrait by Sir Wylie Greer. The retreating hair is now grey, his skilful hands are on his knees, the once sharp eyes now dreamy, but as he sits perhaps he sees in his mind's eye somebody in trouble in some distant spot awaiting his aid. The portrait, the gift of his widow, now hangs in the Academy of Medicine in Toronto. Dr. Abraham Groves died on December 8, 1935. He left it to others to make the claim for him of the earliest planned removal of the appendix for appendicitis. The following are publications of Dr. Abraham Groves as confirmed in the library of the Academy of Medicine, Toronto. The list is almost certainly incomplete because of the vagaries and frequent absence of the indices in early Canadian medical publications.

In the Canada Lancet:
A case of ovariotomy for cyst **6:** 345, 1874
A case of ovariotomy **7:** 192, 1875
A case of double ovariotomy **10:** 195, 1878
Renal calculi **16:** 360, 1885
Vaginal hysterectomy with abdominal ovariotomy **22:** 6, 1889
Immediate closure of the wound after suprapubic cystotomy **25:** 42, 1892
X-Rays in diseased structures **36:** 463, 1903
The diagnosis and treatment of tuberculous peritonitis **37:** 42, 1903
Notes from the Royal Alexandra Hospital, thyroidectomy **41:** 534, 1907
Pyloroplasty **43:** 34, 1909

In the Canadian Medical and Surgical Journal:
Abstract of paper on empyema **12:** 696, 1884
On prostatectomy **15:** 715, 1887

In the Canadian Medical Association Journal:
Evolution of surgery as I have seen it in my own practice **12:** 527, 1922
Rupture of the bladder **13:** 319, 1923
Fractures of the clavicle acromium process and surgical neck of the scapula **14:** 318, 1924

In the Canadian Practitioner and Review:
The radical cure of hernia **29:** 68, 1904

Other papers reprinted in "All in the Day's Work":
Notes on the prevention of disease 1893
Appendiceal disease—Read before the Grey-Bruce Medical Association 1903
The Diagnosis and treatment of tuberculous peritonitis—Read before the Canadian Medical Association 1903
Intra-abdominal anastomosis — Read before the St. Thomas Medical Society 1904
The standardization of hospitals—Read before the Ontario Hospital Association Undated

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RÉSUMÉ

La première intervention pour ablation de l'appendice enflammé après diagnostic exact de la maladie a été faite en 1883 par Abraham Groves dans une ferme près de Fergus en Ontario.

Le Dr Abraham Groves naquit en 1847; il était le fils d'un Irlandais arrivé en 1825 au Canada. Il fit ses études primaires et secondaires dans les écoles publiques de Fergus. Il devint ensuite étudiant en médecine à l'Université de Toronto et reçut son doctorat en 1871. Il eut parmi ses maîtres J. H. Richardson, W. T. Aiken et James Bovell. Il se lia d'amitié avec William Osler qui devint plus tard professeur à l'Université McGill.

Il s'installa comme omnipraticien à Fergus en Avril 1871: il a décrit lui-même ses premières années de pratique dans le livre qu'il écrivit plus tard "All in the Day's Work". Les occasions de travail chirurgical ne manquaient pas: ainsi le Dr Groves eut à pratiquer une transfusion d'urgence en se servant d'une simple poire en caoutchouc, qui fut couronnée de succès. Nombre d'opérations urgentes durent être pratiquées dans des conditions de fortune dans des cabanes ou dans des fermes: on trouvait l'eau dans le puits; les seaux à lait servaient de bassins, la table de cuisine de table d'opération. Bien que les théories de Lister ou de Pasteur n'aient pas encore reçu pleine créance à cette époque, on prenait cependant des précautions de propreté: la région opératoire, les mains du chirurgien et les instruments étaient soigneusement nettoyés. Abraham Groves utilisait un très petit nombre d'instruments: après l'incision de la peau par exemple, le bistouri était mis de côté et les dissections s'effectuaient avec les doigts; la fameuse appendicectomie fut faite seulement avec une aiguille de Hagedorn; souvent, lorsqu'un instrument spécial devenait indispensable, il arrivait qu'il soit fabriqué sur place par le forgeron le plus proche.

Le Dr Groves fonda un hôpital à Fergus: le "Royal Alexandra Hospital," qui devint plus tard propriété de la ville de Fergus, et qui porte aujourd'hui le nom de "Abraham Groves Memorial and Community Hospital".

Les travaux qui s'y effectuèrent étaient à la pointe du progrès pour l'époque: le Dr Groves fit un grand nombre de communications et de publications dans les journaux médicaux, plus particulièrement dans le *Canada Lancet*. Il écrivit de plus son livre.

Il se retira après soixante ans de pratique et de services rendus à la communauté. Il mourut le 8 Décembre 1935, à l'âge de 88 ans.

PEPYS UNDERGOES SURGERY FOR THE STONE*

"Mr. Pepys's operation was performed by Dr. Thomas Hollier of St. Thomas's Hospital; but as was the custom, it was performed in a private home, not at the hospital which was too dangerous and too mean a place for an operation upon a private person of means. Hollier had made a good reputation as a lithotomist, having 'cut' 30 persons for the stone without a single mortality. The Pepyses were fortunate to obtain the services of so capable and so lucky a surgeon.

"He prepared his patient by giving him 'a soothing draught of liquorice, marshmellow, cinnamon milk, rose water, and the white of eggs.' It is hard to know whether this prescription concocted by Dr. James Moleyn, of St. Bartholomew's Hospital, was useful either as a sedative or a digestive, but Pepys did endure the agony of vivisection and recovered to live out his long life, which was somewhat lucky, for Hollier lost his next four patients and gave up the operation.

"Very proud of his stone, Pepys kept it in a bottle to show to any guests who might have the bad fortune to inquire of his operation, or to mention a pain in the bladder region. An even more unfortunate consequence of the operation was that the patient's spermatic duct was injured and to his great sorrow he was thereafter sterile."

*ABERNETHY, C.: *Bull. Am. Coll. Surgeons*, 46: 79, 1961.

ORIGINAL ARTICLES

ENTEROCELE AND PROLAPSE OF THE VAGINAL VAULT*

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and D. E. R. TOWNSEND, M.D., C.M., *Montreal*

THE FEMALE reproductive tract is subject to many forms of relaxation and prolapse. The majority of these would appear to be the result of the trauma of childbirth. A much smaller group is not associated with parturition or its effects and is considered by many to be the result of some congenital weakness. In addition to these types, there are those which may follow various surgical procedures, both vaginal and abdominal. These latter cases are the source of considerable concern to the operating gynecologist, as they are usually attributed by the patient to some faulty technique, or lack of knowledge on the part of the operator. In some instances this may be a justifiable criticism.

The close association of the conditions variously known as hernia of the pouch of Douglas, prolapse of the vaginal vault, prolapse of the cervical stump, posterior vaginal prolapse, posterior enterocele, or enterocele, prompts us to direct our considerations to the etiology, diagnosis and management of these abnormalities. The term "high rectocele", formerly used in describing these conditions, is obviously a misnomer.

HISTORY

Sir Astley Cooper in his classical work on hernia, published in 1804, illustrated a typical enterocele. The earliest reference to this lesion in the literature is in a paper by Thomas[1] in 1885, in which he notes that the condition was first described in 1736 by Garengeot. In 1909 Marion[2] described an abdominal operation designed to obliterate the pouch of Douglas. In 1916 Hartman[3] described a vaginal technique for cure of this condition. Ward,[4] in 1922, recommended the vaginal approach for cure of enterocele which is still largely used.

Since that time important contributions have been made to our knowledge by the recorded experiences of Phaneuf,[5-7] Read,[8, 9] and others. Minor modifications of the original vaginal technique have been proposed[10, 11] as well as methods for use with[12] or following[13] vaginal hysterectomy.

ETIOLOGY

Anatomical Considerations

In 1899 Cunéo and Veau[14] published the results of their anatomical studies and proved that the rectovaginal septum was composed of ventral and dorsal peritoneum fused into a single structure. Their observations, although not uniformly accepted, have now been amply confirmed by Uhlenhuth and his associates.[15-17] They demonstrated that the peritoneal pouch lying between the rectum and the genital organs in the fetus, extends down to the level of the perineal body and as term approaches, there is a gradual fusion of the dorsal and ventral walls of the pouch in a cephalic direction. The extent of fusion varies in individuals and accounts for the differences in depths of the cul-de-sac observed in adult life. They also noted that there is usually a transverse puckering of the peritoneum in the region of this fusion and that the fused septum is more adherent to the fascia about the vagina than that about the rectum. There are thus three distinct layers of tissue lying between the muscular layers of the vagina and the rectum, perivaginal fascia, rectovaginal septum and perirectal fascia.

Thus, on a purely anatomical basis, there is a potential avenue for herniation in every female pelvis, and the clinical entity of enterocele represents either an opening up of the fused portion of the rectovaginal septum, or the occupancy of a pre-existing space by abdominal contents which make

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*From the Department of Obstetrics and Gynecology, The Montreal General Hospital.
Presented at the Regional Meeting, Royal College of Physicians and Surgeons of Canada, London, Ont., November 15, 1960.

the condition symptomatic. The latter might well be dependent on the length of the mesentery or on generalized visceroptosis as is seen in the asthenic type of patient.

Parturition

The effect of childbirth *per se* on the production of these herniations may be debatable. However, most modern gynecologists believe that the trauma of delivery, especially a difficult operative delivery, may cause a defect in the upper portion of the rectovaginal septum with a resultant outpouching behind the cervix and between the uterosacral ligaments. This defect may be enlarged by the expulsive efforts of subsequent deliveries. Indirectly, through causing uterovaginal prolapse, with which most enteroceles are associated, parturition may result in an increase in an already present developmental defect. Read[9] divides the condition into two types — "traction" and "pulsion" enteroceles. Those associated with uterovaginal prolapse as the primary condition he would term the "traction" type.

Intra-abdominal Pressure

Malpas[18] states that "an increase in intra-abdominal pressure and volume is an important secondary determinant of prolapse". He terms it secondary because he believes that a primary cause must already be present in order that prolapse may result. We are entirely in agreement with this concept. There are two factors concerned, the temporary rise in pressure caused by an increase in abdominal volume from ascites, tumour, obesity or by lifting and straining, and the permanent changes which take place in the parietal peritoneum to compensate for this increase. The intra-abdominal pressure is thus returned to normal by the production of some herniation. Thus the "pulsion" type of enterocele results.

Idiopathic Tissue Weakness

It is clinically accepted that in many cases of prolapse there appears to be some inherent weakness in the supporting structures of the pelvis. In the cases of nulliparous prolapse it may be the most important cause. Tissue weakness is a very complex subject that is not adequately understood. It is clearly evident in certain patients during labour and delivery, when "poor tissue tone" frequently increases the hazards. It is also seen in association with debilitating or metabolic diseases, such as anemia and diabetes. It may be responsible for some cases of enterocele or vaginal vault prolapse following hysterectomy.

Inversion of the vagina after hysterectomy is usually not caused by the operation. More often there is already some supporting weakness in the vagina which is not recognized preoperatively. At times, a large uterus may even minimize the vaginal weakness and removal of the organ renders it apparent.

Vaginal prolapse is said to be more common after vaginal hysterectomy than after abdominal hysterectomy. This opinion is debatable when one considers that many more vaginal than abdominal hysterectomies are performed in the presence of varying degrees of pre-existent prolapse.

Vaginal prolapse is conceded to be more common after subtotal hysterectomies than after the total type. This is probably due to the fact that there is less supportive scar about a retained cervix than about the empty vault, and to the fact that a cervical stump acts as a leading focal point for inversion of the tubular vagina.

Diagnosis

In general two main types of enterocele are recognized. The rare "congenital" type, in which there is an elongated, narrow sac extending down between the rectum and vagina as far as the perineal body, is usually bounded above by a narrow opening and represents the failure of fusion of the peritoneal pouch into the rectovaginal septum as already discussed.

The other common type is the so-called "acquired" type, usually believed to be the result of the trauma of delivery. It frequently has a broader neck at its upper end and is often overlooked as it is associated with a more evident condition of uterovaginal prolapse.

When either of these herniations contains

small bowel, the diagnosis is much easier because the presence of a reducible swelling in this location cannot be mistaken. In many cases diagnosis is extremely difficult, especially when the enterocele is small and there is any degree of rectocele present.

Combined rectovaginal examination, using the index fingers of the two hands, will often clarify the diagnosis. Although it is awkward and rather unpleasant for the patient, if this examination is done with the patient standing facing the examiner it is usually easy to determine the presence of a bulging mass protruding into the rectovaginal septum above the rectocele, as shown in Figs. 1, 2 and 3.

The symptoms of enterocele are those of pelvic pressure, bearing down, lack of support and perineal insecurity. Although these symptoms may also be present with most degrees of uterovaginal prolapse, one should be suspicious of enterocele when the symptoms seem out of proportion to the degree of anatomical distortion.

All too frequently the condition remains unrecognized even at operation for rectocele, plastic repairs, prolapse and vaginal hysterectomy. Continuance of the complaints of pelvic pressure when the patient is first allowed out of bed suggests the possibility that an enterocele has been overlooked. In such cases, after vaginal hysterectomy, the presence of an enterocele usually terminates as a complete prolapse of the vaginal vault.

In all patients subjected to plastic repairs, the possible presence of an enterocele must be constantly entertained. Careful attention to this detail will do much to prevent the occurrence of so-called postoperative "recurrent" enterocele. The major proportion of these result rather from the "overlooked" enterocele.

The diagnosis of complete inversion of the vagina or prolapse of the cervical stump is simple and is usually made by a mere inspection of the area. These patients have a great deal of discomfort especially when attempts at voiding or defecation are made. Many require manual replacement of the inverted vagina to succeed in these functions. Lesser degrees of prolapse of the vaginal vault will frequently account for those vague symptoms of pelvic pressure

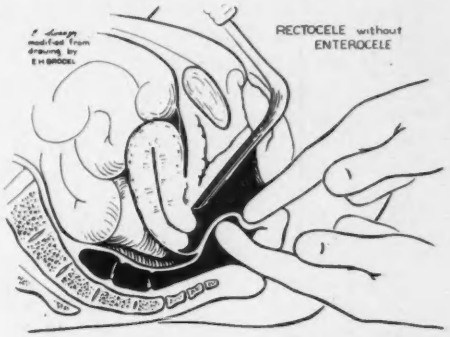


Fig. 1.—Diagnosis of enterocele by combined rectovaginal examination in the dorsal position with an assistant retracting anterior vaginal wall; demonstration of rectocele without enterocele.

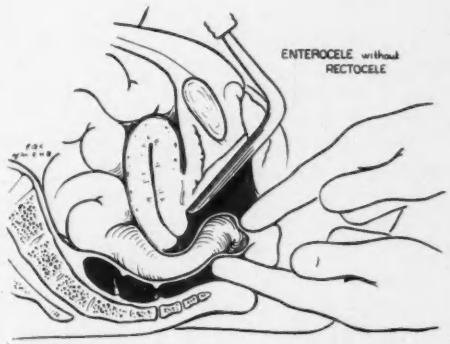


Fig. 2.—Demonstration of enterocele without rectocele by combined rectovaginal examination in the dorsal position.

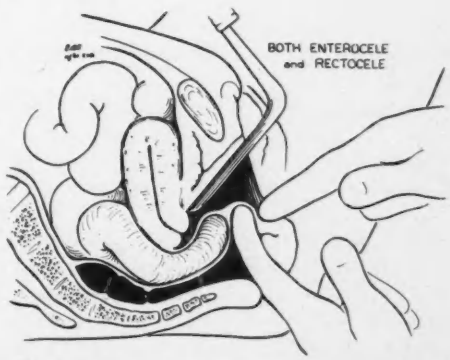


Fig. 3.—Demonstration of both enterocele and rectocele in association, by the combined rectovaginal method.

and bearing down for which no anatomical cause is evident. This is especially true when the patient is examined in the dorsal position. It is important to examine all such patients in the erect posture before excluding this lesion as the cause of such discomfort.

Treatment

In many instances it is possible to prevent the subsequent development of enterocele and vaginal vault prolapse by strict adherence to proper selection of the operative procedure for any individual patient. At abdomino-pelvic operations it is sometimes quite evident that a congenitally deep cul-de-sac or actual hernial sac exists. In these cases, obliteration of the area, either by purse-string catgut sutures in the peritoneum or by interrupted silk in the uterosacral ligaments, will prevent its later appearance.

It is also important to estimate the degree of uterovaginal prolapse that is present in all patients requiring abdominal hysterectomy for any reason. Neglect of this will often lead to an increase in relaxation following hysterectomy and a worsening of an already present minor prolapse.

There are usually three procedures mentioned in the literature for surgical correction of enterocele. The first two to be described were both carried out by the abdominal approach; that of Marion[2] and the Moschowitz method[19] are used primarily to correct rectal prolapse, but are also recommended for vaginal prolapse. The other method is that of Ward,[4] which was performed vaginally and, with some minor modifications, remains the present standard procedure.

In addition, in older patients and for the correction of recurrences after other operations, the Le Fort colpocleisis has been used on occasions with varying degrees of success.

The vaginal approach is the method of choice by gynecologists in the majority of cases. The general principles of treatment are those applicable to hernia. The sac must be exposed, mobilized, opened, the contents replaced, the sac ligated or firmly closed and the redundant part excised. The hernial opening must then be supported by a line of sutures in the margins of the uterosacral ligaments, and approximation of the cardinal ligaments. It is also important to continue this line of support by approximation of the perirectal fascia out to the perineal body.

It cannot be too strongly emphasized that in all vaginal reparative operations one must be constantly aware of the possible presence of an enterocele. Upon occasions it is necessary to open the cul-de-sac before one can rule out its presence. Once discovered it is of primary importance to carefully dissect the entire sac well up to its base, freeing the peritoneum in its complete circumference. It must then be closed completely at that level, either by interrupted chromic catgut sutures or by a purse-string suture of similar type. These are best placed from within the lumen of the sac and very often it is safer to use more than one layer, or more than one purse-string to effect a cure. The redundant portion of the sac should always be completely excised up to the closed end.

In cases of prolapse with enterocele that are being treated by the Manchester technique, or in other instances where the uterus is not removed, the purse-string suture should be anchored to the posterior surface of the upper cervix, and the uterosacral ligaments should be joined together in the midline over the occluded sac.

In vaginal hysterectomy with the uterus removed, before closure of the peritoneum is made it is most important to grasp the posterior fold of peritoneum and by traction downward and backward to closely examine the depth of the cul-de-sac by palpation and direct vision. In opening the posterior pouch it is usual to keep the incision close to the cervix to avoid the rectum and by so doing it is sometimes possible to completely overlook a sizeable enterocele.

The usual technique for vaginal hysterectomy in our series has been that of Campbell,[20] in which the uterosacral ligaments are sutured forward to the pubocervical fascia on their respective sides. The edges of the ligaments are further brought together by interrupted sutures from before backwards to complete the support of the

pelvic floor and occlude the possible enterocele. A purse-string suture, used to close the peritoneum, is also tied about the two uterosacral ligaments in their new position to obliterate dead space above the ligaments.

The most difficult of all cases on which to procure a satisfactory result is that of prolapse of the vaginal vault after abdominal or vaginal hysterectomy, or after an otherwise successful operation for cystocele and rectocele. It is usual in these cases to find a higher incidence of medical complications, cardiac disease, hypertension, diabetes and bronchitis. Although the same general principles for repair are required, it is in these cases that the abdominal approach may offer some advantage. This is especially true if a functional vagina is necessary, as the vaginal approach in such a case uniformly results in a shortening of the vagina.

Clinical Data

We have reviewed a series of 72 cases of enterocele, prolapse of the vaginal vault and cervical stump seen on our service at The Montreal General Hospital over the past five years. These patients have been

TABLE I.—The Montreal General Hospital 1955 - 1959

Gynecological admissions	8241
Abdominal operations	2173
Vaginal operations	696
Dilatations and curettages (minor)	3031

treated by 15 members of the attending and resident staffs. The immediate results have been satisfactory, with only two recurrences in these cases, but we are quite aware that the follow-up period is too short for final assessment, and judging from our experience with the late results of former similar cases, some of the immediate "cures" will eventually experience recurrences. We estimate the recurrence rate in these repairs to be from 5% to 8%.

During the period 1955-59, among 8241 admissions to the gynecology service, there were 423 cases of birth trauma of all kinds. Of these, 48 (11.3%) were classified as enterocele, 16 (3.8%) as prolapse of the vaginal vault and 8 (1.9%) as prolapse of the cervical stump. The combined number of 72 cases comprised 17% of the total cases of birth trauma.

TABLE II.—Birth Trauma 1955 - 1959

	No. of patients	*Percentage*
All types	423	
Enterocele	48	11.3
Prolapse vaginal vault	16	3.8
Prolapse cervical stump	8	1.9
	72	17.0

TABLE III.—Age Incidence

Average age: 58.9 years (37-82)

	No. of patients	*Percentage*
30 - 40 years	2	2.8
40 - 50 "	16	22.2
50 - 60 "	16	22.2
60 - 70 "	29	40.3
Over 70 "	9	12.5

The ages of these patients ranged from 37 to 82 years, with an average age of 58.9 years. The largest group (40.3%) occurred in the seventh decade with 75% of the patients falling into the postmenopausal age group. It is usual to expect an increase in vaginal relaxations after the menopause when general tissue tone becomes poor and associated systemic disease is more common.

TABLE IV.—Parity

	No. of patients	*Percentage*
Nulliparous	5	7
Parous	67	93
Para. 2 or more	56	77
Complicated delivery	17	25.3

Only five patients were nulliparous and they all had enteroceles. Three of these were virginal unmarried women with typical congenital-type narrow sacs extending well down into the rectovaginal septum. Seventy-seven per cent of the parous patients had had more than two full-term children, the maximum number of deliveries being 10. The high incidence of these conditions following pregnancy and the trauma of delivery is further emphasized by the fact that over 25% of the patients

had recorded complicated labours and deliveries.

One of the most interesting aspects of the study was the high incidence of pelvic-abdominal operations which preceded the occurrence of cul-de-sac herniation. Of the total group of patients, 54 (75%) had had 65 previous pelvic operations, almost half of these operations having been performed at the Montreal General Hospital. Sixty per cent of the operations were performed by an abdominal approach and the remainder by vaginal approach.

TABLE V.—Previous Pelvic Operations

		Percentage
Yes	54	75
No	18	25
At the Montreal General Hospital	29	44.6
At other hospitals	36	55.4
Total	65	

TABLE VI.—Abdominal Operations

Total abdominal hysterectomy	20
Subtotal hysterectomy	11
Suspension and fixation	7
Other	1
Total	39

TABLE VII.—Vaginal Operations

Plastic repair	10
Vaginal hysterectomy and repair	15
Watkins	1
Total	26

The time lapse from the original operation to the repair of the cul-de-sac herniation was variable. In patients with enteroceles the extremes were four months to 40 years; in those with prolapsed vaginal vaults the time lapse ranged from one to 36 years, and in those with prolapsed cervical stumps it was from six to 28 years.

TABLE VIII.—Elapsed Time Postoperative

Enterocele	4 months to 40 years
	5—within 1 year
	11—within 5 years
Prolapse vaginal vault	1 year to 36 years
	4—within 1 year
	5—within 5 years
Prolapse cervical stump	6 years to 28 years

TABLE IX.—Enterocele

		Percentage
Congenital	3	6
Acquired	45	94
Enterocele only	5	70.8
" + Rectocele	13	
" + Cysto-rectocele	16	
" + Prolapse	8	29.2
" + Procidentia	6	

More detailed study of the entire series was carried out with reference to the three groups already mentioned. Of the enteroceles only three were of the true congenital type, although the percentage of the acquired cases that were the result of a congenital weakness can only be the subject of speculation. Almost 71% of these were associated with the vaginal relaxations of cystocele and rectocele. The remainder were found in patients with partial or complete uterine prolapse.

TABLE X.—Enterocele

Treatment	
Repair enterocele + vaginal hysterectomy	13
Repair enterocele + anterior-posterior repair	18
Repair enterocele + posterior repair	14
Repair enterocele only	2
No treatment	1

The management of these patients varied with the associated conditions that were present at the time. In each case the enterocele was repaired as a separate entity in addition to the other operation performed and the procedures were almost equally divided between vaginal hysterectomy, anterior and posterior colporrhaphy and posterior colporrhaphy. One patient was treated by pessary support because she also suffered from leukemia, diabetes and heart disease.

The results in this group of patients were good, with only one recorded recurrence. We realize that the period of follow-up has been much too short to consider that they have all been permanently cured. The presence of a shortening of the vagina would seem to be the result of effecting a cure as it was recorded in the follow-up examination of at least 25%. Although this finding

TABLE XI.—ENTEROCELE

Results	
Cured ?	46
Recurred	1
Not treated	1

was not recorded in an additional 30%, it is reasonable to estimate that at least half of these had comparable results. We conclude that it occurs in about 40% of cases and of course, in many elderly patients, it is of little importance.

In the 16 cases recorded as prolapse of the vaginal vault, 10 followed total hysterectomy and five followed vaginal removal

TABLE XII.—PROLAPSE VAGINAL VAULT

16 cases	
Followed total hysterectomy (abdominal) (7 - 32 years)	10
Followed vaginal hysterectomy	5
Followed (1) Suspension	
(2) Subtotal hysterectomy	
(3) Anterior-posterior repair + stump	1

of the uterus. It was a late complication in the abdominal group; no case occurred within seven years of the primary operation and the average was 12 years. All cases of prolapse following vaginal hysterectomy were recognized within five years of the first operation, four of them within the first year. We believe that in these cases insufficient vaginal vault tissue was removed in an effort to preserve a deep, functional vagina. The presence of diabetes in three of these patients may have influenced heal-

TABLE XIII.—PROLAPSE VAGINAL VAULT

Alone	1
With rectocele	1
With cysto-rectocele	9
With cystocele	3
With enterocele	2

ing, although every effort was made in all cases to control hyperglycemia. Fourteen of these patients remain cured; two were not treated. Twelve were treated by vaginal repair, one by a Le Fort operation and one by abdominal repair.

The latter patient appears in all three groups, as she ran the gamut of four operations over a period of 10 years before

TABLE XIV.—PROLAPSE VAGINAL VAULT

Treatment	
Vaginal repair	12
Abdominal repair	1
Le Fort	1
None	2
Result—cured ?	14

eventual cure. She had had a uterine suspension performed at the age of 37 years for prolapse, a subtotal hysterectomy at the age of 42, an anterior and posterior colporrhaphy with removal of the cervical stump at 46 years of age and finally an abdominal repair of enterocele and prolapse of the vaginal vault at the age of 47. At present, four years after the latter operation, she remains cured with a good functional vagina.

The cases of prolapsed cervical stump all followed subtotal hysterectomy, only one having an associated vaginal plastic performed at the same time. The presence

TABLE XV.—PROLAPSE CERVICAL STUMP

8 cases	
All followed subtotal hysterectomy (6 - 28 years)	
All with cysto-rectocele	
All treated—Vaginal plastic + excision stump	
Result—cured ? 7	Recurred 1

of some degree of cysto-rectocele at the time of the hysterectomy was quite probable, as it was found in all of the cases. Contrary to reports in the literature, in our series, prolapse of the cervical stump was a relatively late occurrence, appearing from six to 28 years postoperatively. All of these cases were repaired vaginally with excision of the cervical stump. Only one recurred, the case already detailed above.

CONCLUSIONS

The common occurrence of enterocele and hernia of the cul-de-sac demands the attention of all gynecologists.

Although the majority of cases seem to occur as a result of the trauma of parturition, a congenital abnormality may be the primary cause.

The diagnosis and the basis of treatment is discussed.

The clinical data on 72 cases are recorded.

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Résumé

Le tractus génital féminin est le siège de nombreuses formes de prolapsus; le plus souvent, ceux-ci sont la suite du traumatisme de l'accouchement; un groupe beaucoup moins important est sans relation avec la parturition et semble consécutif à des facteurs de faiblesse congénitale; enfin, toutes sortes d'interventions chirurgicales, abdominales ou vaginales peuvent jouer un rôle dans leur apparition.

De nombreux termes sont utilisés pour désigner des malformations très semblables: hernie du cul-de-sac de Douglas, prolapsus de la voûte vaginale, prolapsus du moignon cervical, prolapsus vaginal postérieur, entérocèle postérieure. Des considérations anatomiques peuvent aider à comprendre l'étiologie de ces troubles: le septum recto-vaginal est formé par la fusion de lames péritonéales ventrale et dorsale sur une hauteur qui peut varier, ce qui explique les différences observées cliniquement dans la profondeur du cul-de-sac de Douglas. Il existe donc un point faible dans le bassin féminin, prédisposant aux hernies.

L'accouchement lui-même, spécialement les accouchements difficiles ou compliqués de manœuvres opératoires, peut provoquer un affaiblissement de la partie supérieure de la cloison recto-vaginale: le résultat sera une hernie en arrière du col, entre les ligaments utéro-sacrés. Ce processus sera aggravé par les accouchements ultérieurs.

De plus, toute augmentation anormale de la pression intra-abdominal (épanchement d'ascite, tumeurs, obésité) aboutira au même résultat.

Dans certain cas, il faut admettre la possibilité d'une faiblesse congénitale des tissus: ce concept est encore mal compris, mais il explique les prolapsus que l'on observe chez les nullipares. Le renversement du canal vaginal après hystérectomie relève de la même cause: cette inversion n'est pas due à l'intervention, mais à une faiblesse préexistante et souvent masquée par la présence d'un gros utérus.

Du point de vue diagnostique on s'accorde à reconnaître un type "congénital" et un type "acquis". Les symptômes de l'entérocèle sont une sensation de pression dans le bassin et de "manque de support". Bien trop fréquemment le diagnostic n'est fait que lors de l'intervention.

Pour ce qui est du traitement, il est essentiel d'adapter la technique opératoire à chaque cas particulier. Plusieurs procédés ont été proposés; dans l'opinion de l'auteur, le meilleur est une modification de l'intervention par voie vaginale de Ward. Les principes généraux à respecter sont ceux de la cure de hernie: il faut exposer le sac, l'ouvrir, réduire le contenu et le ligaturer. L'orifice herniaire sera refermé par une ligne de sutures dans les bords des ligaments utéro-sacrés, et par le rapprochement des ligaments cardinaux.

Le présent article se termine par la revue de 72 cas d'entérocèle et de prolapsus soignés à l'Hôpital Général de Montréal pendant les cinq dernières années. Les résultats de cette étude statistique sont donnés sous forme de tableaux.

ACUTE SURGICAL DISEASE OF THE ABDOMEN COMPLICATING PREGNANCY*

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A 23-YEAR-OLD girl in the eighth month of her third pregnancy reports to the emergency ward at 8 o'clock in the morning complaining of moderately severe abdominal pain. Her previous pregnancies have been uneventful and her past history noncontributory; in fact, she has always enjoyed good health. Review of the chart, in retrospect, reveals a rather cursory initial history and examination, as a result of which no definitive diagnosis is reached. In this history one recognizes an unwritten but implied belief that a pregnant woman has some mysterious right to abdominal pain and vomiting which defies medical explanation. Eight hours later the patient is complaining of severe pain and examination reveals an elevated pulse rate and marked abdominal tenderness. Concern for her welfare is now apparent from the frequent progress notes and from the consultant's report that a diagnosis of abruptio placenta must be considered. A further delay of six hours follows during which emergency blood and radiological examinations are obtained. During this interval the pulse rate rises to 120 per minute, the temperature to 102°F. and the abdomen becomes slightly distended, with generalized rigidity, tenderness, rebound tenderness and absent bowel sounds. At laparotomy two hours later the abdomen is reported to be "full of pus" and the gangrenous appendix to have perforated at its base.

While such a case may be a relatively uncommon surgical experience, it is none the less a factual one and doubtless accounts for the healthy respect with which the medical profession views acute surgical disease of the abdomen complicating pregnancy.

The present review began in the four active treatment hospitals in the city of Edmonton as a 10-year survey of acute surgical disease of the abdomen during pregnancy. Unfortunately, complete coding of such cases was not carried out in all hospitals throughout this entire period. The cases therefore have been drawn from 23 cumulative hospital years and represent 59,758 confinements (Table I).

TABLE I.—CASES REVIEWED

Hospitals (Edmonton, Alta.)	*Period*	*Years*	*Number of deliveries*
University of Alberta	1950-1959	10	16,243
Royal Alexandra	1955-1959	5	26,322
Edmonton General	1955-1959	5	10,767
Misericordia	1957-1959	3	6,426
		23	59,758

ACUTE APPENDICITIS

Acute appendicitis was the commonest extragenital cause of an acute surgical abdomen developing during pregnancy (Table II). In this series of 98 patients with surgical disease of the abdomen, almost all of whom underwent operation, 50 of the operative procedures were carried out with a preoperative diagnosis of acute appendicitis.

TABLE II.—EXTRAGENITAL ACUTE SURGICAL DISEASE OF THE ABDOMEN DURING PREGNANCY

(98 Cases)

Appendicitis (clinical diagnosis)	50
Cholecystitis	20
Hernia	7
Bowel obstruction	6
Pancreatitis	4
Ureteric calculi	4
Acute malignancy	2
Peptic ulcer	2
Ulcerative colitis	1
Acquired hypertrophic pyloric stenosis	1
Perforation of the ileum	1

The analysis of these cases as to the final pathological diagnosis is of considerable

*Presented at the Annual Meeting of the Manitoba Division of the Canadian Medical Association, September 26, 1960, and the Annual Meeting of the Saskatchewan Division of the Canadian Medical Association, October 21, 1960.

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TABLE III.—Appendectomy during Pregnancy
Pathological Diagnosis

(50 Cases)

Acute suppurative or gangrenous appendicitis		30
(a) Without perforation	25	
(b) With perforation	5	
Subacute appendicitis		5
Acute or subacute periappendicitis		4
Normal		11
(a) Normal	9	
(b) Obliterative fibrosis	2	

interest (Table III). In 30 cases acute suppurative, ulcerative or gangrenous appendicitis was present, and in five of these frank perforation had occurred with generalized peritonitis from which *Escherichia coli* was cultured. In 11 cases the appendix must be looked upon as being innocent as the cause of abdominal pain, the pathological report describing a normal appendix in nine and obliterative fibrosis in two.

The incidence of acute appendicitis complicating pregnancy has been given as 1 in 1200 cases by Philpott[13] on a basis of 7147 obstetrical cases studied at the Royal Victoria Hospital in Montreal. Hamlin, Bartlett and Smith[5] reported an incidence of 1 in 2300 cases based on their observations on 92,772 deliveries at the Boston Lying-In Hospital. The incidence in the present series is 1 in 1992.

The average age of this group of patients with acute appendicitis was 22.4 years and their parity is illustrated in Fig. 1. It is of interest to note that 70% of the cases occurred during the first two pregnancies.

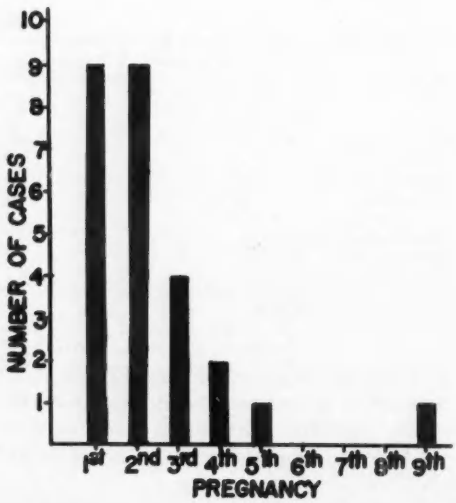


Fig. 1.—Incidence of acute appendicitis according to stage of pregnancy in 26 cases.

Diagnosis

The diagnosis of acute appendicitis occurring during the first and second trimesters of pregnancy appeared to offer no great difficulty in this series. The typical story of vague periumbilical or epigastric pain, later increasing in intensity, localizing in the right lower quadrant and associated with anorexia, nausea and often vomiting, appears on the charts with monotonous regularity. The physical findings too were not dissimilar from those observed in the non-pregnant state. Right lower quadrant tenderness was constant and almost invariably associated with rebound tenderness. Rovsing's sign was frequently recorded as being positive. It is said that muscle-guarding is minimal or absent in these cases because of the stretching of the abdominal wall that accompanies pregnancy.[5] In the 11 charts in this series in which this finding was specifically mentioned it was minimal or absent in five and moderate to marked in six.

TABLE IV.—Appendectomy during Pregnancy
Onset of Disease

	First Trimester	*Second Trimester*	*Third Trimester*
Acute appendicitis			
Unperforated	9	13	3
Perforated			5
Subacute appendicitis	2	2	1
Periappendicitis	2	1	1
Normal	5	5	1
Total	18	21	11

While the diagnosis of acute appendicitis was invariably made during the course of the initial hospital examination when this disease occurred during the first two trimesters, this was not the case in the last trimester of pregnancy. It is usually stated that acute appendicitis occurs most frequently in the first six months of gestation,[7-14] but in this series there was little indication of such a predilection (Table IV). One feature of acute appendicitis

occurring in the last trimester is however obvious, namely, the frequency with which it tends to progress to perforation and generalized peritonitis before therapy is initiated. There were eight cases of acute appendicitis in the third trimester, of which five had perforated and, in this series at least, all cases of perforated appendicitis occurred in the last trimester. A possible reason for this is apparent when one analyzes the charts, for while the average interval between the onset of symptoms and operation was 20.6 hours in the unperforated cases, it was invariably over 48 hours in those with perforation. It would be grossly unfair to imply, however, that this delay was invariably due to procrastination on the part of the attending physician, for in three of the five cases the patient presumably misinterpreted the symptoms as being a normal accompaniment of late pregnancy and reported only when peritonitis was established. The difficulty that the physician may encounter in establishing a diagnosis of appendicitis close to term is illustrated by the case from this series that was used to introduce this presentation.

The white blood cell count and differential count are said to be of little help in the diagnosis of appendicitis in pregnancy because of the physiological leukocytosis that accompanies pregnancy.[8] While we would agree that a white blood cell count is seldom necessary in order to establish a diagnosis of appendicitis in any patient, there was a definite trend towards higher counts in the patients with acute appendicitis as compared with the "normal" controls in this series. The average count in 20 cases of acute appendicitis in which it was recorded was 16,500 per c.mm. with an average of 87% polymorphonuclear leukocytes. The average leukocyte count in the 11 "normal" cases was 12,800 per c.mm. with an average of 80% polymorphonuclear leukocytes. In none of the "normal" cases did the count exceed 16,500, while counts in seven of the acute appendicitis cases exceeded this figure. We do not feel, however, that the white blood cell count is a necessary or even a particularly useful aid to the diagnosis of appendicitis in pregnancy.

Treatment

There is general agreement[4, 5, 9, 14] that signs and symptoms suggestive of acute appendicitis make early exploration mandatory in the pregnant female. Many pathophysiological changes inherent in the pregnant state have been recorded as favouring a more rapidly progressive and fulminating course for intra-abdominal inflammatory disease associated with pregnancy.[4, 14] The mortality and morbidity of acute appendicitis during pregnancy arises from procrastination and delay.

In this series the McBurney incision was favoured and was used in half of the 50 cases. We personally favour this incision, particularly where the diagnosis is reasonably assured. The operative notes often indicated that "the incision was made at a slightly higher level than normal because of the complicating pregnancy", but the anatomical position of the appendix was not recorded in sufficient detail to confirm the study of Baer, Reis and Arens[1] who reported that the appendix is displaced upwards and laterally and undergoes a counter-clockwise rotation with the progress of pregnancy.

Prognosis

There was no maternal mortality in this series, although the increased morbidity of appendiceal perforation is indicated by the average hospital stay of 14.7 days for this group as compared with an average of 6.2 days for the cases of acute unperforated appendicitis.

The fetal loss in the entire series is summarized in Table V. The two patients with acute unperforated appendicitis who aborted in the first trimester did so on the twelfth and fourteenth postoperative days after they had been discharged. Neither had aborted during previous pregnancies. The single patient who aborted in the second trimester did so on the sixth postoperative day and had spontaneously aborted two of three previous pregnancies.

Three of the five patients with acute perforated appendicitis went into premature labour, but the fetus was salvaged in two who had reached the seventh and eighth

TABLE V.—APPENDECTOMY DURING PREGNANCY
SUMMARY OF FETAL LOSS

	First trimester		*Second trimester*		*Third trimester*		
	Number	*Aborted*	*Number*	*Aborted*	*Number*	*Premature labour*	*Fetal loss*
Acute appendicitis							
Unperforated	9	2	13	1	3	—	—
Perforated	—	—	—	—	5	3	1
Subacute appendicitis	2	1	2	—	1	—	—
Periappendicitis	2	—	1	—	1	1	—
Normal	5	1	5	—	1	—	—
Total	18	4	21	1	11	4	1

months of gestation. In the other case, fetal loss occurred at seven months' gestation.

Fetal loss also occurred in two other patients undergoing appendectomy in the first trimester.

In the "normal appendix" case the record is of interest. This 19-year-old primipara was operated upon at six weeks' gestation for severe right lower quadrant pain of two-and-a-half days' duration with typical physical findings of a right lower quadrant inflammatory process. At operation the appendix was normal but the right ovary contained a cyst "the size of a tangerine". The patient was known to be pregnant and the possibility of a cystic corpus luteum was entertained by the competent gynecologist in attendance. However, he considered the ovarian mass to be "most certainly" a follicular cyst and it was removed. The pathological report indicated that it was a luteal cyst of the ovary. The patient began to have lower abdominal cramps the day after operation and passed a complete sac three days later.

In the single case of acute periappendicitis occurring at eight months' gestation, operation precipitated labour but a viable fetus was obtained.

CHOLECYSTIC DISEASE

It was impossible from the material available for this study to determine the incidence of cholecystic disease occurring during pregnancy. Many, if not most, cases so diagnosed with or without radiological confirmation of disease were not coded in a manner to make them consistently accessible for analysis if conservative management was the only form of therapy instituted. It is generally agreed in the literature[5, 14] that a conservative approach to acute cholecystitis occurring during pregnancy is desirable and that operation should be reserved for those patients failing to respond to such a regime, or for those who manifest complications such as perforation, empyema, common bile duct obstruction or concomitant pancreatitis. In chronic recurrent cholecystitis, interval postpartum cholecystectomy is favoured.

Diagnosis

The diagnosis of cholecystic disease occurring during pregnancy appears to present no special features apart from those commonly seen in the non-pregnant female.

In so far as surgery directed to the biliary tract during pregnancy is concerned, Stone and Folsome[14] make a strong plea for conservatism on the basis of:

1) The technical difficulties of biliary surgery in the presence of a gravid uterus.

2) The excellence of the prognosis for subsidence of the acute attack with conservative management.

3) The high incidence of termination of pregnancy associated with the combination of intra-abdominal suppuration and biliary tract surgery.

In view of the widespread acceptance of the philosophy of conservatism in this condition, we were somewhat surprised to find that in 20 cases of the present series operation was carried out for inflammatory disease of the gallbladder during pregnancy. The patients were older than those who underwent appendectomy, the average age being 30.25 years. The relation of parity is also interesting and is compared in Fig. 2 with that of the group undergoing appendectomy.

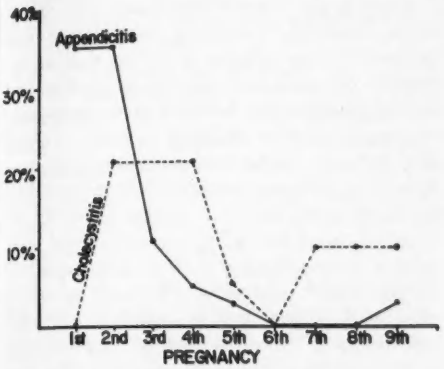


Fig. 2.—Relation of parity to disease.

Indications for Operation

The indications for surgery in this series appear to be somewhat broader than those described in the literature, and are summarized in Table VI. It is probable that the number of biliary tract operations does not accurately reflect the normal incidence relative to the number of deliveries in these same hospitals over the same period of time, for only eight of 20 patients were from Edmonton and would normally be confined in a hospital in that city.

TABLE VI.—Biliary Tract Surgery during Pregnancy

(20 Cases)

Average Age: 30.25 Years

Indications for Operation	
Increasing recurrences; moderately acute on admission	10
Increasing recurrences; not acute on admission	6
Moderately acute	2
Acute	2

The assessment of the acuteness of the disease, as recorded in Table VI, is a clinical one. Pathologically, 16 cases were classified as chronic cholecystitis, one as subacute cholecystitis and one as acute cholecystitis; all of these were associated with cholelithiasis. In one patient a normal gallbladder without stones was removed. In the remaining patient, in whom the clinical and the operative diagnosis was acute cholecystitis, cholecystostomy only was carried out and no tissue was available for study. Two patients had slight jaundice on admission to hospital.

Treatment

The operative procedures carried out in these 20 cases are summarized in Table VII. The magnitude of many of these procedures is of interest in connection with the prognosis for fetal survival in the group.

TABLE VII.—Biliary Tract Surgery during Pregnancy

Procedures	
Cholecystostomy	1
Cholecystectomy	7
Cholecystectomy and appendectomy	6
Cholecystectomy and exploration common duct	5
Cholecystectomy, appendectomy and exploration common duct	1

Prognosis

In this group of 20 patients, one remains lost to follow-up. There was no maternal mortality. Sixteen patients continued to full-term delivery of a viable healthy child. Fetal loss occurred in three cases (Table VIII). In one of these, a 21-year-old multipara in the third month of gestation, abortion occurred on the day of cholecystectomy. Her previous pregnancies had all gone to term. The other two patients were both multipara who had previously aborted on one occasion, and following the biliary tract surgery, abortion occurred after an interval of two months in one and after three months in the other. In these two cases there is, therefore, reasonable doubt as to the causal relationship between the surgery and the fetal loss.

TABLE III.—Biliary Tract Surgery during Pregnancy
Summary of Fetal Loss

First trimester		*Second trimester*		*Third trimester*	
Number	*Aborted*	*Number*	*Aborted*	*Number*	*Premature labour*
8	2	8	1	3	0

Hernia

In this series herniorrhaphy was performed on seven occasions during pregnancy. Four of the hernias were inguinal and three umbilical. An apparent paradox exists in the relation of inguinal hernia to pregnancy.[2, 10] Some hernias, manifest in the pregravid period, disappear as pregnancy progresses, presumably due to displacement of the viscera normally present in the sac, away from its ostium, with enlargement of the gravid uterus. In other cases defects which were not apparent in the pregravid state became manifest during pregnancy only to regress following delivery. Laval and Theodore[10] draw attention to the fact that not all inguinal masses occurring during pregnancy are hernias, and mention among the other causes of such a mass the occurrence of engorgement of veins in the inguinal canal. One such case, treated surgically, was noted in obtaining material for this review but has not been included since no urgent indication for intervention existed.

There is general agreement in the literature that the presence of an inguinal or umbilical hernia is not an indication for operation in the pregnant woman.[2, 10] The patient should be made aware of the potentially serious complications of the lesion, should be watched with care, and subjected to immediate operation should incarceration or strangulation supervene. Only one patient in this series fulfilled these requirements for operation. This was the case of a 38-year-old multipara of four months' gestation, who noted an exquisitely tender mass at the umbilicus of four days' duration, which was associated with vomiting on the day of admission. At operation infarcted omentum in an umbilical hernial sac was resected and a Mayo-type repair was effected. Pregnancy continued uneventfully to term.

In the remaining six cases the indication for operation appears to have been increasing local pain and discomfort, although in no case was this severe. Five of these patients went on to full-term normal delivery, while the sixth, a 24-year-old multipara in her sixth month of gestation, aborted three days after repair of a small umbilical hernia.

During the period of this study in one of these hospitals (U.A.H.) there were nine patients whose charts recorded that an inguinal or umbilical hernia was observed during pregnancy, but in whom pregnancy was permitted to continue to term. Following delivery, after an average interval of five days, surgical repair was accomplished in these cases without complication. However, in spite of the apparent facility with which herniorrhaphy may be accomplished in the early postpartum period, it is our observation that better tissue is available for repair if complete puerperal involution is allowed to occur before operation is performed.

Intestinal Obstruction

Intestinal obstruction is fortunately a rare complication of pregnancy[2, 6, 10, 12] for it carries a grave prognosis. Hansen[6] in 1941 stated that among the cases reported in the English literature there was a maternal mortality of 31.25% and a fetal mortality of 57%. More recent publications would suggest that this gloomy prognosis has been much improved in the last 20 years, but no extensive recent review has come to hand.

Statistics for the province of Alberta gathered by the Maternal Mortality Committee of the Alberta Division of the Canadian Medical Association record 186 maternal deaths in 327,809 confinements in the 10-year period from 1950 to 1959. Deaths due to extragenital acute surgical disease of the abdomen accounted for only four of the 186 maternal deaths and, of these, three were due to small bowel obstruction. The one remaining maternal death was a case of perforated appendicitis with peritonitis. None of these maternal deaths occurred in the series reported here.

Diagnosis

The diagnosis of intestinal obstruction occurring during pregnancy appears to constitute the major hazard in the successful management of the patient. Colicky abdominal pain, abdominal distension, vomiting and constipation are complaints common to both pregnancy and intestinal obstruction. Since intestinal obstruction com-

TABLE IX.—Intestinal Obstruction during Pregnancy

(6 Cases)

Average Age: 30 Years
Pregnancy: First to Seventh
Gestation: 4 to 6½ Months: Average 5.6 Months

Etiology	
Adhesive bands	3
Small bowel intussusception	1
Pregnancy ileus	1
Abdominal pregnancy	1

monly occurs in the latter part of gestation it is not surprising that these symptoms are often initially dismissed as indicating the onset of early labour. This tetrad demands careful consideration at any stage in pregnancy. A past history of an intra-abdominal surgical procedure should increase one's suspicion, as approximately one-half of the cases of intestinal obstruction are due to adhesive bands (Table IX).[3] Suspicion once aroused should lead to a careful interrogation of the patient with regard to the rapidity with which distension developed, the nature of the vomitus and the occurrence of passage of flatus. Examination of the abdomen must be painstaking and detailed, and special attention should be paid to the presence of high-pitched bowel sounds and signs of peritoneal irritation. Where the diagnosis is in doubt reassessment at frequent intervals is essential. The relation of the cramping pain to peristaltic rushes on the one hand or to palpable uterine contractions on the other may assist in this difficult differentiation. Plain roentgenographs of the abdomen, supine and erect, will prove to be the most useful laboratory aid to diagnosis.

Treatment

The management of intestinal obstruction in pregnancy in no way differs from that recommended in the non-pregnant patient. The frequency with which resection of non-viable bowel is found necessary and the difficulty encountered in the preoperative separation of a simple from a strangulating obstruction in the pregnant patient does, however, necessitate a more aggressive surgical approach than in the non-gravid person.

Prognosis

In this series there was no maternal mortality. Four of the six patients went to full term and delivered a normal child in spite of the fact that in two cases gangrenous bowel required resection at the time of laparotomy. The patient with pregnancy ileus[10] underwent two operations within a week of admission, subsequently contracted a wound infection and went into premature labour three weeks after her second operation, delivering a 3 lb. 4 oz. viable fetus at seven months' gestation. The only fetal loss occurred in the patient with abdominal pregnancy in which the fetus was removed at laparotomy during the fifth month of gestation. This was the only case in this series in which conservative measures proved effective in controlling the obstruction.

Acute Pancreatitis

The incidence of acute pancreatitis complicating pregnancy, as distinct from that in the puerperium, is recorded by Langmade and Edmundson[11] as 1:5798 pregnancies. These authors presented six such cases and reviewed the 53 acceptable cases in the literature to that date (1951). In the present series there were four cases, an incidence of 1:14,939 pregnancies.

In view of the paucity of recorded cases of this disease the essential features of our four cases will be reviewed.

Case 1.—R.A.H. No. M12068.—This 29-year-old woman was admitted to hospital on June 16, 1954, at 31 weeks' gestation in her first pregnancy. She had an 18-hour history of epigastric pain without radiation, which was associated with nausea and vomiting. Examination revealed only epigastric tenderness. Five hours after admission she complained of continuous right abdominal and loin pain with evidence on examination of intense peritoneal irritation in the right lower quadrant.

Emergency laparotomy was performed with a preoperative diagnosis of appendicitis, but acute hemorrhagic pancreatitis was readily apparent. No surgical procedure was carried out. Her postoperative serum amylase level was 200 Somogyi units.

After an initially stormy convalescence she progressed uneventfully to term and delivered a healthy child.

In the subsequent six months she suffered four similar attacks of short duration, and cholecystectomy and removal of three stones from the common bile duct was accomplished without difficulty. Her subsequent course was uneventful.

Case 2.—U.A.H. No. 89379.—This 28-year-old woman, gravida VI, whose last menstrual period was on October 31, 1955, was first admitted to hospital on January 29, 1956, with acute epigastric pain radiating to the back. Cholecystogram, barium meal and serum amylase level were all normal and she was discharged undiagnosed.

The patient was readmitted with recurrent symptoms one month later at which time the serum amylase level was 914 Somogyi units. She responded to a conservative regimen.

Readmission with an acute recurrence on April 18, 1956, associated with marked progressive abdominal tenderness, led to cholecystojejunostomy the following day. Her convalescence was uneventful.

On August 8, 1956, full-term delivery of a normal child occurred.

On December 4, 1956, cholecystectomy, splenectomy and excision of the body and tail of the pancreas was performed as three recurrent attacks had occurred in the interval since delivery.

On December 12, 1958, at which time she entered hospital for treatment of a protruded intervertebral disc, the patient reported that she had had no further episodes of abdominal pain.

Case 3.—U.A.H. No. 163661.—This 25-year-old woman, gravida I, whose last menstrual period was on May 3, 1957, entered hospital on November 13, 1957, with a two-day history of epigastric pain and vomiting. The white blood cell count was 19,250 per c.mm., total serum bilirubin 5.31 mg. % and serum amylase level 940 Somogyi units. On a conservative regimen for acute pancreatitis her symptoms subsided only to recur in a more severe form in one week. On November 21, 1957, cholecystostomy was performed. Convalescence was uneventful.

On January 28, 1958, she was readmitted with recurrent symptoms and a serum amylase of 1000 Somogyi units, but she rapidly responded to a conservative regimen.

On February 5, 1958, she was delivered of a healthy child at term. On February 14, 1958, cholecystectomy and exploration of the common duct was carried out and after a stormy course she was discharged on her eighteenth postoperative day.

Case 4.—U.A.H. No. 184005.—This 21-year-old woman, gravida IV, was first admitted on August 4, 1959, at which time she was not pregnant. She gave a history of five severe attacks of epigastric and back pain over the course of the previous three years, two of which occurred in the postpartum period. Her most recent attack, which preceded admission by seven weeks, was associated with jaundice. Examination revealed a firm smooth non-tender mass in the mid epigastrium. Serum amylase studies were normal but she manifested mild diabetes mellitus. She was discharged on a conservative regimen with a diagnosis of subsiding acute pancreatitis and possible pseudocyst formation.

On Octover 18, 1959, she was readmitted. She had had no menstrual period since the birth of her last child and it was now established that she was three months' pregnant. Shortly before this admission she had suffered a recurrent attack of severe abdominal pain. At laparotomy on November 3, 1959, the diagnosis of relapsing acute pancreatitis was confirmed and cholecystectomy, after operative cholangiography, was performed. Convalescence was uneventful and she was returned to the care of her own doctor with the suggestion that pregnancy be interrupted and sterilization be carried out. This was subsequently accomplished uneventfully.

It is apparent from a study of these four cases that the triad of epigastric pain, nausea and vomiting, especially if associated with epigastric tenderness, occurring during pregnancy demands consideration of acute pancreatitis as the possible etiological factor. The diagnosis can be confirmed relatively simply in the early stages of the disease by the demonstration of elevated serum amylase levels. A conservative regimen similar to that recommended for acute pancreatitis in the non-pregnant person is the treatment of choice. The necessity of performing operative decompression of the biliary tract during pregnancy in two of the four patients in this series would suggest that conservative measures alone are frequently inadequate. There was no maternal mortality in this small group and it is surprising that the only fetal loss that occurred was the result of planned thera-

peutic interruption of pregnancy recommended because of the recurrent episodes of pancreatitis. All three patients permitted to go to term had subsequent definitive surgical procedures on the biliary tract and in both of those in whom a significant interval occurred between delivery and the second procedure, recurrent acute attacks occurred in the puerperium.

Miscellaneous Conditions

Eleven additional cases of acute abdominal disease complicating pregnancy were encountered in this series. There were four patients with ureteric calculi, all of whom presented with typical symptoms and physical findings and required surgical removal of the stone for relief. Additional patients were discharged from hospital undelivered with a diagnosis of ureteric colic due to stone during the period reviewed, but the diagnoses must be considered unconfirmed as the stones were never recovered, no operation was performed and intravenous pyelography studies were often equivocal. In the cases included, stone removal was accomplished by transurethral bucket extraction in three and by operative ureterolithotomy in one. All patients went on to normal full-term delivery.

There were two cases of duodenal ulcer. One was classed as acute because of the severity of the subjective complaint of pain, although there was no evidence of perforation. The other case was so classified because recurrent hemorrhage resulted in serious consideration being given to the advisability of emergency gastrectomy. However neither of these women was operated upon as the symptoms were satisfactorily controlled on a medical regimen and the patients went on to normal full-term deliveries.

There were two cases of intra-abdominal malignancies that presented as acute emergencies. One 34-year-old multipara presented with symptoms suggestive of acute cholecystitis with jaundice at five months' gestation. At laparotomy adenocarcinoma of the ampulla of Vater was found and pancreaticoduodenectomy was performed. This patient aborted the day after operation and died of extensive metastatic disease four months later. The second patient with malignant disease was a 36-year-old multipara who presented with acute obstruction of the left colon at two months' gestation. Biopsy of the sigmoid lesion at the time of colostomy and hysterotomy confirmed the diagnosis of adenocarcinoma. The patient expired three months after operation.

One interesting case of perforation of the ileum was encountered. This 18-year-old primapara had undergone appendectomy and excision of a Meckel's diverticulum at two weeks' gestation, although pregnancy was unsuspected at that time. Two months later while in hospital for treatment of hyperemesis gravidarum she had an acute episode of abdominal pain with physical findings suggestive of peritonitis. Immediate laparotomy revealed perforation of an ulcer of the ileum situated at the point of surgical closure of the Meckel's diverticulum. Her subsequent convalescence was uneventful. There were no further episodes of hyperemesis and she delivered a normal infant at full term.

There was one patient who suffered an acute recurrence of ulcerative colitis during her first pregnancy after a remission that had lasted six years. She responded well to psychotherapy, bed rest, diet and cortisone and subsequently carried on to term.

The last patient presented with vomiting and abdominal pain suggestive of cholecystic disease. Operation was undertaken because of the persistence of, rather than the severity of, the symptoms. The operative findings were consistent with a diagnosis of acquired hypertrophic pyloric stenosis of the adult and a Ramstedt procedure was performed with relief of symptoms. Full-term delivery was uneventful.

Summary and Conclusions

Acute appendicitis is the commonest extragenital cause of acute abdominal disease ocurring during pregnancy. The tetrad of abdominal pain, anorexia, nausea and possibly vomiting in the pregnant woman requires immediate and careful assessment in all trimesters if serious sequelae are to be avoided. The diagnosis, relatively simple in the first two trimesters, is progressively

more difficult as term approaches and procrastination at this time is fraught with the danger of perforation, maternal morbidity (or even mortality), premature labour and fetal loss. Abortion is more commonly associated with appendectomy in the first trimester than in the second, but a diagnosis of acute appendicitis is an indication for immediate laparotomy regardless of the stage of gestation, an indication more urgent in the pregnant than in the non-pregnant state. It is often said that laparotomy and removal of a normal appendix carries no risk to either mother or child during pregnancy. This statement might be considered to be substantiated in this study as long as removal of the appendix is not accompanied by the removal of the corpus luteum during the first trimester.

Cholecystic disease occurring during pregnancy is usually considered to be ideally treated by conservative measures and we would agree with this attitude. It would appear from this series, however, that surgery directed at the biliary tract does not carry with it as grave a prognosis with regard to maternal and fetal morbidity and mortality as the literature would suggest.

Intestinal obstruction tends to occur during the latter months of pregnancy and carries with it a grave prognosis. This would appear to be largely due to delay in diagnosis through confusion of the symptomatology with that of early labour. Surgical intervention is more urgently indicated in intestinal obstruction in the pregnant than in the non-gravid patient.

Acute pancreatitis occurs during pregnancy with sufficient frequency that this diagnosis must be considered in any patient presenting with epigastric pain, nausea and vomiting. The treatment is primarily medical but biliary decompression may be necessary during pregnancy for control of the disease. Recurrent attacks in the postpartum period frequently necessitate further definitive surgery.

Acknowledgment

It is a privilege for me to acknowledge the assistance that I received from the doctors and record librarians in the city of Edmonton in connection with this study and to record my appreciation.

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Résumé

L'appendicite aiguë est le principal syndrome abdominal aigu que l'on voit survenir en même temps que la grossesse. L'ensemble des quatre symptômes suivants: douleur abdominale, anorexie, nausée et vomissements chez une femme enceinte exige un examen immédiat et attentif pour éviter des séquelles sérieuses. Le diagnostic, relativement simple dans les deux premiers trimestres, devient peu à peu plus délicat au fur et à mesure que l'on approche du terme. Tout retard ou toute méconnaissance de la maladie aggrave considérablement le pronostic pour la mère et l'enfant à cause du danger de perforation. L'avortement en relation avec l'appendicectomie se produit plus fréquemment dans le premier trimestre que dans le second; cependant, un diagnostic d'appendicite

aiguë est une indication de laparotomie immédiate, sans se préoccuper du stade de la grossesse.

D'une façon générale, l'appendicectomie semble être sans danger sérieux pour la mère ni pour l'enfant: ceci n'est vrai qu'à la condition formelle que le corps jaune de l'ovaire ne soit pas touché pendant l'opération.

Les cholécystites pendant la grossesse doivent être traitées de façon conservative. Il faut cependant reconnaître qu'il ressort des études statistiques présentées dans cet article que la chirurgie sur le tractus biliaire n'est pas aussi dangereuse qu'on pourrait le croire à première vue.

L'obstruction intestinale tend à survenir dans les derniers mois. Le pronostic en est très grave en soi; mais souvent, la difficulté de faire le diagnostic différentiel entre l'obstruction et le début du travail assombrit encore la situation. L'intervention chirurgicale est hautement indiquée, plus même que chez des malades en dehors de l'état de grossesse.

Enfin, il faut penser en présence d'une malade qui souffre de douleurs dans la région épigastrique, de nausées et de vomissements, à la pancréatite aiguë. Le traitement est avant tout médical, mais l'intervention peut être nécessaire pour décomprimer les voies biliaires ou encore, après la grossesse par des attaques récurrentes.

RUPTURE OF THE LIVER IN CHILDREN: A 34-YEAR REVIEW AT THE HOSPITAL FOR SICK CHILDREN, TORONTO

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Introduction

Rupture of the liver following injury to the abdomen or chest presents a serious threat to life. The ligaments of this heavy, solid organ prevent its escape from the forces involved and thus tears may occur near their attachments. Such an injury will result in subcapsular hematoma or a laceration through the capsule and parenchyma with resultant blood and bile peritonitis. However, the most serious threat to life is hemorrhage. Bacterial invasion of the damaged liver with advancing gangrene resulted in a high mortality until the importance of large doses of antibiotics was recognized.[14]

Injuries of the liver may be classified as direct (penetrating) and indirect (non-penetrating or blunt trauma). Direct injuries were by far the commonest in the literature reviewed. In Sparkman's series,[19] 92% were of the penetrating type and in Madding's and DeBakey's series 80% were of this type.[12, 16] Stab wounds and missiles were the common offenders. Blunt trauma is the usual means of injury to the liver in children.

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Diagnosis

In contrast to penetrating injuries, damage to the liver by blunt trauma is difficult to assess and therefore has a higher mortality. Associated injuries and the presence of shock add further delay in diagnosis. However, in a conscious patient upper abdominal pain plus shoulder-tip pain is suggestive, together with respiratory grunting or difficulty in breathing. A ruptured spleen or diaphragm may result in similar symptoms. Both bile and blood are peritoneal irritants and cause early generalized abdominal pain, tenderness and rigidity. A tender palpable liver may indicate a large subcapsular hematoma.

Shock on admission is a poor prognostic sign. In DeBakey's series, there was an 80% mortality in patients presenting with systolic blood pressures of from 0-50 mm. Hg.[16] If the diagnosis is rupture of the liver, operation is mandatory.[1, 12]

Treatment

Large reserves of blood must be available and should be given in adequate amounts, because the treatment is a major surgical operation on a sick patient in whom the hepatic synthesis of fibrinogen and prothrombin may be impaired.[7, 8] In Madding's series,[12] 62% of the non-operative group died. All authorities agree on the general principles of stopping the hemor-

rhage; removal of devitalized liver tissue, repair of the defect and drainage of the peritoneal cavity.[9] Madding states that 91% of his patients had spontaneous hemostasis.[12] The use of hemostatic agents may be necessary, but some may act as a foreign body and cause further trouble. The use of Ivalon has been recommended and this material has been employed successfully in liver surgery.[6, 11]

Complications are much more frequent in the undrained cases.[9, 10, 12, 16] These are associated with a higher incidence of subphrenic abscess, bile peritonitis and hemobilia.[9, 20]

Postoperative care involves adequate hydration with intravenous electrolyte solutions and blood if necessary. Large doses of antibiotics are also recommended. Decompression of the gastrointestinal tract with nasogastric suction may be advisable.

Mortality

Mortality rates are difficult to compare due to a great number of variables. Among 339 patients, Madding reported a 9.7% mortality,[13] whereas, when the liver was the only organ involved, there was a 6.4% death rate.[12] The majority of these injuries were of the penetrating type, which has a much better outlook. In the closed type caused by blunt injury, the mortality is higher. DeBakey reported a 71% fatality in his series,[16] and attributed this to the high percentage of patients with associated injuries.

Eye Complications

Involvement of the retina is an interesting complication of rupture of the liver which has been reported by Cashell.[5] In patients with this lesion the fundi showed exudate and hemorrhage with thinning and attenuation of the arterioles. The cause of these findings is in doubt but they are said to be due to fat embolism, with toxemia and vasomotor spasm playing a part. Most patients recover but some may go on to develop optic atrophy. This is probably a rare complication and in most instances the patient may not complain of symptoms related to it.

Rupture of the Liver in the Newborn

About 1% of neonatal deaths are said to be due to liver rupture.[17, 18] This lesion ranks next to intracranial injuries as the second commonest form of fatal trauma in the newborn. The first record of recovery from neonatal rupture of the liver was reported by Rogers[18] in 1933 and the second successful case was reported in 1946.[2] In 1951 Arden reported on six newborn infants with rupture of the liver, four of whom survived.[3, 15] Life-saving measures include immediate blood transfusion and administration of vitamin K.

Contributing and predisposing factors are a large baby, a premature infant, traumatic forceps delivery and excessively energetic slapping of the baby at birth. It is important for the obstetrician and pediatrician to be aware of this injury. The value of laparotomy in the newborn is still debatable and it appears that blood transfusion is the most valuable therapy at the present time.[4]

The Hospital for Sick Children Series

From 1924 to 1958 there were 58 cases of known or suspected rupture of the liver at The Hospital for Sick Children, Toronto. In the suspected or non-operated cases which survived, the diagnosis was based on finding some of the following: an appropriately located external injury; shifting dullness; shoulder-tip pain; decreased hemoglobin; increased liver size; and abnormal liver function test values.

There were 32 deaths (56% mortality) among the 58 patients reviewed. Of the 26 survivors, a positive diagnosis was made in nine patients at operation.

There were seven cases of rupture of the liver in the newborn in this series, all of whom died within a few hours of admission. Five of these infants were premature. In all of these patients there were associated injuries or congenital anomalies. Five had pulmonary complications; two had intracranial hemorrhage and one had total anomalous pulmonary venous drainage.

Among the remaining 51 patients there were 25 deaths (49%) as a result of various accidents (Table I). The contributing

TABLE I.—Cause of Liver Injury

	Patients	Deaths	Mortality Percentage
Automobile accidents	33	21	64
Fall from a bicycle	5	0	0
Fall from height	5	2	40
Sleigh-riding	3	1	33
Stepped on by a cow	1	1	100
Struck by a swing	1	0	0
Football injury (knee in abdomen)	1	0	0
Struck by falling object	2	0	0
Birth injury	7	7	100
Total	58	32	55

lethal factor was due to one or more associated injuries. Hepatic injury when it occurred alone resulted in a mortality of 8%. The sites of associated injuries were the abdomen (89%), chest (83%) and central nervous system (69%) (Table II). Of

TABLE II.—Types of Associated Injury

Injury	Patients	Deaths	Mortality Percentage
Chest:	20	16	83
with fractured ribs	12	10	83
without fractured ribs	8	6	75
Central nervous system	16	11	69
Fractures:	13	7	54
Long bones	10	5	50
Pelvis	3	2	67
Intra-abdominal	18	16	89
Spleen	5	4	80
Adrenal	4	4	100
Bowel	3	3	100
Mesentery	3	2	67
Major vessel	1	1	100
Pancreas	1	1	100
Kidney	1	1	100
Urinary bladder	1	1	100
Diaphragm	2	2	100
Retroperitoneal hematoma	4	3	75

the 26 survivors only eight had associated injuries (31%), whereas in the fatal group of 25 there were 21 associated injuries (84%) (Table III).

TABLE III.—Frequency of Associated Injuries

	Total number of cases	Associated injuries No. of cases	Percentage
Survivors	26	8	31
Deaths	25	21	84

Age and Sex

Excluding the neonatal cases, the age group most commonly affected was the 5-10 year group, with boys predominating three to one.

Signs and Symptoms

Abdominal tenderness and rigidity were commonly recorded, as were shifting dullness, shoulder-tip pain and enlargement of the liver. The presence of shock on admission was a poor prognostic sign and was associated with a 67% fatality. Twenty-four of the 25 patients who died were in shock on admission. Only 13 showed signs of shock in the group of 26 survivors.

Other findings suggestive of liver damage were a fall in hemoglobin and altered liver function tests. These were recorded in a few cases (Table IV). The majority of the tests were performed on the patients who were not operated upon.

TABLE IV.—Laboratory Results in Patients with Ruptured Liver

	Patients	Deaths	Survivors
Fall in hemoglobin	8	1	7
Altered liver function tests	6	0	6

Surgical Management

In the group of 51 patients only 12 were operated upon. In the remaining 39 there was a mortality rate of 56% (22 patients). Six of these died in the first hour after admission and the remainder died within the first 24 hours.

There were three deaths in the group of 12 surgical patients, two of whom died within 24 hours.

TABLE V.—Mortality from Hepatic Injury

	All patients			Excluding patients dying in 1st hour		
Period of study	Number	Deaths	Mortality Percentage	Number	Deaths	Mortality Percentage
1924-1940	20	14	70	15	9	60
1941-1944	4	2	50	4	2	50
1945-1947	4	2	50	4	2	50
1948-1958	23	7	28	22	6	27

TABLE VI.—Operative Procedures

	Patients	*Deaths*	*Survivors*	*Mortality Percentage*
With drainage				
Hemostatic gauze and suture	2	0	2	
Suture only	2	0	2	
Total	4	0	4	0
Without drainage				
Cotton gauze packing	2	2	0	
Suture only	3	1	2	
Hemostatic gauze and suture	1	0	1	
Total	6	3	3	50
Delayed				
Gauze pack	1	0	1	
Drainage only	1	0	1	
Total	2	0	2	0

The mortality rate has been greatly reduced by improved methods of treatment but largely through the availability of blood for transfusion. In the earlier years there were more deaths within the first hour of admission. The deaths among patients who were operated upon all occurred before 1941 (Table V). Thirty-one of the 51 patients were transfused. The amounts of blood varied, but only in the past 12 years were adequate volumes given.

A summary of the methods of treating the 12 surgical patients is outlined in Table VI. This simply emphasizes the value of draining the peritoneal cavity. It is interesting to note that all three operative deaths occurred in the group in which no drainage was used. Three patients survived without drainage but in two of these there were delayed complications which required drainage of pocketed bile in one and the treatment of intestinal obstruction in the other.

In the group in which the abdominal cavity was drained (4 cases) there were no complications (Table VII).

TABLE VII.—Complications in Operative Cases

With drainage		4 cases
Complications		None
Without drainage	6 cases and 2 delayed cases	
Complications		10
Subphrenic abscess	2	
Bile peritonitis	2	
Adhesions	2	
Bowel obstruction	1	
Necrosis of liver	2	
Wound infection	1	
Deaths		3

Summary and Conclusions

A review of the literature has been presented to emphasize some of the problems in the diagnosis and treatment of rupture of the liver.

Fifty-eight cases of rupture of the liver in neonates, infants and children have been reviewed from the records of The Hospital for Sick Children, Toronto.

Seven cases resulted from trauma at birth; none of these patients survived.

Most of the injuries resulted from automobile accidents (33 patients with 21 deaths).

Associated injuries were common, occurring in 21 of the 25 fatal cases.

More adequate use of blood transfusions, antibiotics and early operation has resulted in a lower mortality rate.

The primary aims of operation are the arrest of hemorrhage and drainage of the peritoneal cavity.

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Résumé

Les blessures du foie à la suite de traumatismes peuvent être classées en deux groupes: les blessures directes ou pénétrantes qui sont de loin les plus fréquentes; les blessures indirectes, qui, du fait de la difficulté de leur diagnostic, ont un pronostic beaucoup plus grave.

Cet article présente une étude d'ensemble de 58 cas de blessures hépatiques traitées à l'Hôpital des Enfants Malades de Toronto de 1924 à 1958. Dans cette série il y a eu 32 morts (56%); il est à noter que chez les 26 survivants le diagnostic pré-opératoire n'avait été fait que dans 9 cas.

Il y eut 7 cas chez des nouveaux-nés, qui succombèrent tous quelques heures après l'admission. Cinq de ces enfants étaient prématurés; tous étaient porteurs de lésions associées ou de malformations congénitales.

Chez les adultes, la mort fut causée le plus souvent par des traumatismes secondaires; lorsque la lésion hépatique était unique, la mortalité tomba à 8%.

La cause de la plupart de ces blessures était l'accident d'automobile. Les symptômes les plus fréquemment rencontrés furent: douleur abdominale et contracture musculaire pariétale; douleurs projetées dans l'épaule; hypertrophie du foie à la palpation. L'état de choc était peu significatif.

Du point de vue thérapeutique, il est important, avant et pendant l'intervention de donner des transfusions en quantité suffisante et des antibiotiques à doses massives. Le drainage de la cavité abdominale permet de lutter contre la péritonite biliaire.

DIAGNOSIS OF CARDIAC ARREST*

"Theoretically, diagnosis of cardiac arrest should not present much difficulty. The recognition of the functionless heart in abdominal and thoracic procedures is easy; its immobility, the absent arterial blood pressure and the change of colour is obvious. Yet the surgeon engrossed in a fine dissection may fail to appreciate for some seconds the cessation of the circulation. It happens quietly, but usually with some warning signs. Fluctuations in heart rate, changing rhythms, multiple extrasystoles, and bouts of tachycardia — particularly ventricular tachycardia — commonly herald the abrupt cessation of the circulation. A direct-writing electrocardiograph or a cardiac monitor screen in the theatre will become before long a basic essential. It alerts all the attendants to the possibility of an abrupt cardiac fatality. On the screen the anesthetist or his assistant can view with accuracy the behaviour of the heart and observe at the same time a continuous record of blood pressure. By such means precious seconds in treatment will be saved. The electroencephalogram provides an even better means of determining an absent heart beat: the loss of alpha, beta and gamma waves occurs within 20-25 seconds of total anoxia of the brain."

*Gilchrist, A. R.: The cardiac patient as a surgical risk, *J. Roy. Coll. Surgeons Edinburgh*, **6:** 159, 1961.

REPORT ON 41 CASES OF RUPTURE OF THE SPLEEN

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THE OBJECT of this paper is to report on 41 cases of rupture of the spleen and to discuss certain aspects of this condition.

The material reported was obtained from the records of the Hamilton General and St. Joseph's Hospitals during the last 10 years. Only patients in whom the diagnosis was established at operation or autopsy are included.

We found that it was not possible to tabulate neatly and completely the clinical and laboratory data because there were so many variable factors. For example, 21 of the patients had multiple injuries, many were unconscious on admission, and some were intoxicated. Of the 21 with multiple injuries, 13 had fractured ribs on the left side, 10 had blood in the urine and 10 had other serious lesions. Possibly the most important variable of all was the amount of time which elapsed between the injury and the onset of significant signs and symptoms.

Bearing this in mind, we found that the following were the most prominent symptoms:

1. Abdominal pain which was usually but not always, in the left upper quadrant.
2. Shoulder pain; in the left shoulder nine times and in both shoulders once.
3. Weakness, fainting, "shock".
4. Nausea and vomiting.

The most prominent abdominal signs were:

1. Tenderness – especially in the left upper quadrant.
2. Rebound pain—usually referred to the left upper quadrant.

Muscle splinting, distension and shifting dullness were not prominent among the recorded signs, but in our own experience distension and moderate resistance are important signs. As will be shown later, the abdominal signs and symptoms were sometimes most misleading.

Evidence of serious hemorrhage was recorded in 28 patients. Fourteen were stated to be in shock and 14 had a hemoglobin of less than 10 g. %.

The white blood cell count was reported in 10 cases. In one it was 6000 per c.mm., in one 8000, in another 11,600 and the rest varied between 16,000 and 32,000 per c.mm.

The time that elapsed between injury and diagnosis was variable. Many patients recovered from the immediate effects of the injury and seemed better. One man actually went back to work for 24 hours and one housewife carried on for 10 days in the belief that she had sustained only a broken rib. McIndoe[1] was one of the first to draw attention to the fact that severe bleeding does not always immediately follow rupture of the spleen. Ten of these patients fit into his category of "delayed rupture" (that is, they went through an asymptomatic period of 48 hours or more), and in 15 others splenectomy was not considered necessary until more than 24 hours after injury.

It seems worth while to describe briefly the course of several of the patients with delayed bleeding, because these are the ones who present most of the difficulties in diagnosis.

CASE 1.—A man, aged 48 years, was admitted to hospital on July 4, 1956, after a motor accident. He was fully conscious and not in shock. There were no abnormal findings in his abdomen. He had 11 fractured ribs on the left side and a fracture of the left tibia.

On July 9 an open reduction of the fractured tibia was performed. On July 13 he suddenly went into shock; his blood pressure dropped to 60/0 mm. Hg. His abdomen appeared distended and there was tenderness in the left upper quadrant. A diagnosis of delayed rupture of the spleen was made and after suitable resuscitation the spleen was removed. Its posterior surface was torn and there was a large amount of blood and clot in the peritoneal cavity. His postoperative course was satisfactory.

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CASE 2.—A girl, aged nine years, had fallen off a fence 48 hours before admission and for an hour had complained of pain in the left upper quadrant. The next day she began having pain in the right lower quadrant with anorexia and nausea. The fall had been forgotten.

On admission her temperature was 102° F., her pulse rate was 100 per minute and her blood pressure was 120/80 mm. Hg. There was considerable tenderness in the right lower quadrant and no distension. Her white blood cell count was 8000 per. c.mm. and her hemoglobin was 12 g. %.

A diagnosis of appendicitis was made and the abdomen was opened through a McBurney incision. A large amount of dark blood was found. The McBurney incision was closed and the ruptured spleen was removed through a paramedian incision. She was discharged from hospital on the tenth postoperative day.

CASE 3.—A 42-year-old man was admitted because of abdominal pain, which was rather widespread but worse in the right lower quadrant, accompanied by nausea and vomiting. The pain started on a Monday. He had consumed a considerable amount of alcohol over the week-end. He gave no history of injury except that he had bumped his chest while swimming during the week-end. The physical signs were rather confusing but in the surgeon's opinion they indicated an acute inflammatory process—most likely acute appendicitis. Again the abdomen was opened through a McBurney incision and again considerable free blood was found. A paramedian incision was made and a ruptured spleen was removed. This procedure was carried out on the same day that the symptoms appeared. There was no postoperative complication.

CASE 4.—A 39-year-old man had fallen 15 ft. from a scaffold and landed on his left side. He complained of pain in the left chest, left upper abdomen and left elbow. On examination, his colour was good; his pulse rate was 100 per minute and his blood pressure was 105/60 mm. Hg. There were no clinical or roentgenological signs in his chest. There was considerable splinting of his abdomen with tenderness, most marked on the left side. There were no bowel sounds. Rebound pain was present. The urine was clear. There was a compound fracture of the olecranon process.

After two hours' observation, the abdominal signs had largely disappeared. The compound fracture was repaired.

The next day his hemoglobin was 75%. Eighteen days later he began to complain of vague abdominal pain. There was slight resistance and tenderness in the left upper quadrant. His hemoglobin was 55%. Radiographs showed elevation of the left diaphragm and a barium meal disclosed that the stomach was displaced slightly to the right by a mass in the left upper quadrant.

After transfusion, the abdomen was opened and a splenectomy was carried out. This was performed with considerable difficulty because the area around the spleen was sealed off by adhesions; the tissues were edematous and the bleeding profuse.

The postoperative course was stormy. On the fifteenth postoperative day a subphrenic abscess was drained, but the patient did not recover completely. He began to cough up purulent sputum. A bronchopleural fistula was demonstrated. Seventy-five days after the splenectomy a second drainage of the subphrenic abscess was undertaken, after which the patient made good progress.

CASE 5.—A 40-year-old woman was involved in a motor accident and was struck in the left lower anterior chest by a gearshift lever. A puncture wound was present in the sixth intercostal space at the anterior axillary line, but there was no rib fracture or pneumothorax. During the next three weeks her hemoglobin sank slowly to 60%. A mass developed in the left upper quadrant. Her temperature remained elevated, the puncture wound became mildly infected and a pleural effusion formed. However, she recovered with aspiration, transfusions and antibiotics. At first, the mass in the abdomen receded, but after four months it began to grow again and gave rise to pain and distress. Six months after the injury this patient was operated upon. A very large cystic hematoma was found between the spleen and stomach. It was plastered to each of these organs, to the splenic flexure and to all adjacent tissues. In removing the spleen and cyst, the blood supply of the splenic flexure was cut off. This part of the colon was exteriorized. Two further procedures were needed to restore the continuity of the bowel and the health of the patient.

PRE-EXISTING SPLENIC DISEASE

Of the 41 ruptured spleens, only one was previously diseased. It belonged to an alcoholic who was involved in a motor accident in another city. At first, the most prominent injuries were several broken ribs on the left side and a large hemothorax. The chest was aspirated and after

six days the patient insisted on returning home. He was readmitted to hospital and in the following days it was noted that (1) the red blood cell count was falling; (2) there was increasing distension of the abdomen, and (3) a mass had appeared in the left upper quadrant. An abdominal paracentesis was performed and bloody fluid was obtained. The spleen was removed 14 days after injury through a left subcostal incision. The spleen was definitely enlarged and lacerated. The liver was not examined. Later, histological examination of the spleen revealed dilated sinusoids, fibrosis and endothelial hyperplasia. Liver tests showed impaired function. It is of interest that this patient was the only one in whom an abdominal paracentesis was performed as a diagnostic test.

During this same period it was found that two patients who were operated upon in the belief that their spleen had been ruptured, did not, in fact, have this injury. One was a young woman who sustained a severe blow to the left upper quadrant and left lower chest in a fall from a haystack. There was marked pain and tenderness in the left upper quadrant with slight guarding. There was no rib fracture. The white blood cell count was 24,000 per c.mm. After three hours' observation, the abdominal signs and symptoms were still present, though there were no definite indications of severe hemorrhage. At operation no intra-abdominal injury was found. Her recovery was uneventful. The other patient was a 5-year-old boy. After being struck by a car he developed increasing pain, tenderness and splinting in the left upper quadrant. His pulse rose and his blood pressure fell. His hemoglobin was 65%. At operation, there was a small retroperitoneal hematoma above the left kidney. He also made a satisfactory recovery.

The postoperative complications of these operations were:

Evisceration	1
Large hematoma in abdominal incision	1
Postoperative pneumonia	1
Subphrenic abscess and bronchopleural fistula	1
Injury to blood supply of splenic flexure	1

There were six deaths in this series of patients.

(1) A woman was injured in a motor accident, sustaining a badly compounded fracture of the left leg which was treated surgically. Twenty-four hours later she collapsed and died. At autopsy a ruptured spleen and massive intra-abdominal hemorrhage were found.

(2) A young farmer suffered a ruptured spleen by a kick from a cow. The spleen was removed but the patient died on the eighth postoperative day. At autopsy it was discovered that he had thrombosis of the splenic and portal veins and extensive infarction of the intestines.

(3) A boy of seven years fell off his bicycle, struck his abdomen, walked home, collapsed and was brought to hospital. He died on the way to the operating room three and one-half hours after his fall.

(4) A man of 31 years, with concussion and a "stove-in" chest, died three days after splenectomy. At postmortem the condition of the abdomen was satisfactory. He had contusion of the heart and massive atelectasis.

(5) and (6) Two men were admitted to hospital unconscious, with cranial and other injuries, including ruptured spleens. These two patients never regained consciousness. The final diagnosis was made at autopsy.

Discussion

There are no specific effects of rupture of the spleen. Hemorrhage is the only serious result of this injury. Although it is not really known whether serious hemorrhage *always* follows rupture of the spleen, it is established that this occurs often enough to be very dangerous, sooner or later.

Concerning the characteristics of the hemorrhage, three types were distinguished in these 41 cases.

(a) Hemorrhage occurring immediately after rupture or within a few hours of it.

(b) Delayed hemorrhage.

(c) Persistent slow bleeding (or possibly repeated short episodes of bleeding).

According to Boyd,[2] the hemorrhage which begins early does so because the capsule of the spleen is ruptured. If there

is a short delay, it is due to contraction of the muscular framework of the spleen or to contraction and retraction of the torn arteries, which is overcome when the blood pressure rises. Boyd postulates that delayed rupture is probably due to the fact that originally only the pulp of the spleen is torn and the capsule remains intact. When the capsule gives way days later, a copious hemorrhage ensues.

Thirdly, we have concluded that in some patients the bleeding was either slow but persistent, or else intermittent, and that this type is characterized by:

(1) falling red blood cell count; (2) increasing distension; (3) development of a mass in the left upper quadrant, and (4) fever.

Obviously, many of these patients presented with a real problem. There was no concern about the choice of operation; it was splenectomy, but in a doubtful case the question to settle was whether or not to perform this operation, particularly in the presence of other major injuries. Boley, McKinnon and Schwartz[3] express one point of view in stating that in children the diagnostic triad of (1) history of trauma; (2) presence of abdominal or shoulder pain, and (3) abdominal tenderness and guarding, especially in the left upper quadrant, are sufficient indications for laparotomy. Gross,[4] on the other hand, states, "Inasmuch as the violence usually produces some contusion of the abdominal wall, the physician often has difficulty in deciding whether the pain and tenderness are due to trauma of the abdominal wall or to injuries of organs within the abdominal cavity. In such a quandary a falling blood count is of great importance in the differential diagnosis."

We do not believe that this study will decide the question. As has been said, there are so many variable factors to consider that probably the surgeon's judgment and past experience will be the strongest influences in making his decision. In any event, one of the first things to do when injury to the spleen is a possibility, is an estimation of the hemoglobin. Of course, such an estimation taken one or even two hours after injury gives no information about the amount of bleeding.

To quote Samson Wright,[5] "It should be stressed again that *immediately* after a hemorrhage the total hemoglobin content of the blood is reduced, but the hemoglobin *percentage* is unchanged". But if one has a base line to begin with, subsequent hemoglobin levels have a real significance.

Secondly, it goes without saying that if a waiting attitude is adopted, the patient must have the closest attention for many days, with repeated hemoglobin estimations.

When days go by before definite evidence is established of hemorrhage and/or hematoma formation, we believe that splenectomy should be performed promptly thereafter, because:

(1) There is real danger of further bleeding.

(2) There is danger of infection.

(3) The operation itself is more difficult and dangerous if undertaken after the hematoma has begun to organize.

One other decision sometimes has to be made. When should a patient who *might* have a ruptured spleen be discharged from hospital? Again, variable factors exist and no rigid rule can be laid down in this respect.

Summary

Forty-one cases of ruptured spleen have been reported, of whom six died. We believe, firstly, that this injury is a treacherous one, because although the only result of rupture is bleeding, yet the time at which the bleeding starts and the amount of bleeding which may occur, are quite unpredictable. Secondly, once the presence of bleeding has been proved, removal of the spleen is advisable.

Acknowledgment

The cases described in this report were under the care of the members of the staff of the departments of surgery at the General Hospital and St. Joseph's Hospital, Hamilton, Ont.

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Résumé

Cet article est un rapport statistique concernant 41 cas de rupture de la rate, établi d'après les archives de l'Hôpital Général de Hamilton et de l'Hôpital St. Joseph de la même ville.

Les cas se sont présentés de façons très diverses: 21 d'entre eux portaient des contusions et des blessures multiples et variées. On peut cependant en dégager les faits suivants, du point de vue symptomatologique: la douleur abdominale existe toujours et elle siège souvent dans l'hypochondre gauche; cette douleur est fréquemment projetée dans une ou deux épaules; état de choc et collapsus; nausées et vomissements.

Le temps écoulé entre l'accident et l'établissement du diagnostic fut très variable: un homme reprit son travail pendant les 24 premières heures et une femme put vaquer à ses occupations régulières pendant une dizaine de jours, croyant avoir simplement une fracture de côte.

L'hémorragie est évidemment le facteur responsable de la gravité de la situation; cette hémorragie n'est pas toujours immédiate, ce qui explique parfois le temps de latence entre l'accident et l'apparition des symptômes. En fait on peut distinguer trois modalités: l'hémorragie immédiate et abondante; l'hémorragie tardive et le saignement lent mais persistant. Il semble que ces différences soient dues à la capsule splénique capable de se contracter grâce à ses fibres musculaires lisses. Dans les hémorragies chroniques, il se fait une diminution du nombre des globules rouges, une masse apparaît dans l'hypochondre gauche et la fièvre s'installe. L'infection de l'hématome est possible, venant compliquer la situation.

Dans tous les cas, le traitement le plus indiqué est la splénectomie, aussitôt que possible. Dans les saignements chroniques, la même intervention reste valable: il y a intérêt à la faire avant que l'hématome n'ait eu le temps de s'organiser.

TRAUMA AND THE LIVING CELL*

"Summarizing the chief directives for winning the battle fought at the cellular level for recovery after trauma, the following technical points are put forward, all aiming at the protection of the life of the cell in severe open injuries.

"Prevent and treat shock which by decreasing the circulation of the damaged tissues leaves them at the mercy of bacteria and reduces further the number of surviving cells.

"Clean the skin and wound with soap and water to eliminate bacteria and foreign bodies.

"Exercise extreme conservatism with the skin, removing only its torn edges, but extirpate dead muscle; and exercise reasonable conservatism in handling bone.

"Only exceptionally close by primary suture of the skin an accidental wound causing severe muscle damage. In good hands this may be done in peace-time injuries; but never in war injuries.

"When the wound is initially left open, to achieve the best drainage of its depths use calico absorbent gauze which maintains the surfaces of the tissues dry.

"Use firm but gentle external pressure as it is essential to the good health of the vascular-connective tissue and the epithelial cells of the wound surfaces.

"Immobilize and protect wounds when necessary with plaster casts or splints, and elastic bandages. These insure healthy vessels and a well drained wound for the first days until it is covered by skin suture or by grafting within one week from infliction of wound.

"Systematically elevate the extremity, in the absence of major vessel injury. This reduces edema, contributes to the normality of cell metabolism and to the satisfactory end result.

"The foregoing are the principles and practice of a method of wound treatment which I promulgated during the Spanish Civil War and later recommended and used during the second World War.

"This method has little in common with that recommended by Dr. H. Winnett Orr for the treatment of established osteomyelitis, with which it was unfortunately confused. Cleaning of the wound, excision of devitalized tissues, and drainage with dry absorbent gauze were meant to prevent infection while Orr designed treatment to control suppurating bone sepsis. When this latter treatment was applied to freshly inflicted wounds and fractures it caused frequent, severe complications and always an offensive smell."

*Trueta, J.: *Bull. Am. Coll. Surgeons*, **46**: 73, 1961.

TRAUMATIC HEMOBILIA

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Massive hemorrhage from the biliary tree, as a complication of closed hepatic injury, was first described by Sandblom[1] who coined the term "traumatic hemobilia". In his review Sandblom quoted Owens, who in 1848 published what is probably the earliest report of such a case.

Incidence

Traumatic hemobilia is very uncommon. Most textbooks fail to mention it. Aird wrote that it occurred rarely, and Sandblom[1] was able to collect histories of nine cases. Sparkman[2] added three more to this list.[3] There are two cases reported to date in the Canadian literature.[7, 8] Two further cases are presented in this paper, one proved and the other based on a diagnosis made in retrospect. Although this is obviously a rare condition, one can infer from the diagnostic difficulties involved that such cases occur much more frequently without the proper diagnosis being made.

Clinical Picture

The majority of the reported cases of traumatic hemobilia were adult males engaged in vigorous activities, but it has been encountered in children as young as three years of age. Closed trauma, usually quite severe, was the cause in all instances. This was commonly associated with the signs of intraperitoneal hemorrhage. The clinical picture usually improved gradually over the first week and in many of the reported cases, including the two reported here, the patients had been discharged from hospital before the onset of biliary hemorrhage, which rarely occurred before two weeks had passed. In a few the first bleeding was delayed for several months.

The biliary hemorrhage mimicked upper gastrointestinal bleeding from any cause. Hematemesis occurred in more than half the patients, all had bloody stools, and all showed anemia if not frank hypovolemic shock.

The biliary obstruction was manifested as biliary colic, obstructive jaundice, or a distended gallbladder. In most of the reported cases biliary colic preceded the hemorrhage and subsided with its onset. The hemobilia was episodic and several patients were able to predict subsequent hemorrhage because of the warning colic. In some cases blood clots, in the form of casts of the biliary tree, had been passed per rectum or vomited.

Diagnosis

With the history of abdominal or lower thoracic trauma, upper gastrointestinal hemorrhage should immediately suggest hemobilia. If associated with the characteristic pain of biliary colic the diagnosis is almost certain. Obstructive jaundice and distension of the gallbladder are confirmatory signs. In spite of many reports to the contrary, we feel that in most instances it is possible to make the diagnosis on purely clinical grounds.

Pathology

The hepatic tear is usually central, with rupture of a fairly large biliary radicle. Sandblom[1] suggested that capsular tears bled intraperitoneally more readily than into the biliary tree, but that once they healed superficially or were sealed by the diaphragm they were converted into central ruptures and could then bleed into a duct. Sparkman[2] felt that the lesion was a cystic cavity periodically communicating with the biliary tree. He postulated the occurrence of an accumulation of bile and blood under pressure in a cystic cavity, which led to lysis of adjacent liver and the release of the fluid into the biliary tree. This relieved the pressure and bleeding stopped, only to recur as the process was repeated.

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Prognosis

The mortality rate in reported cases was 50%. However, many of the fatalities occurred before the general use of blood transfusion, giving a falsely pessimistic viewpoint.

Case Reports

Case 1.—J.B., (No. 442523), an eight-year-old boy, was admited to hospital in September 1959 after a motor vehicle accident. He suffered fractures of the right tibia, fibula and clavicle, as well as an undetermined abdominal injury manifested by diffuse pain and tenderness and voluntary rigidity. With regard to the abdomen, no active surgical treatment was instituted. Improvement was spontaneous and he was discharged 12 days after the accident.

Six days later he was readmitted with the story of severe upper abdominal colicky pain followed by hematemesis and a tarry stool. Upper gastrointestinal roentgenograms were normal. Exploratory laparotomy was performed revealing no cause for the hemorrhage. On the tenth postoperative day a severe hemorrhage was again preceded by upper abdominal pain. Repeat radiological examination was reported as suspicious for duodenal ulcer, and on December 23, 1959, the boy was transferred to The Hospital for Sick Children, Toronto, where he had several more attacks of pain followed by hematemesis. On one occasion his blood pressure fell because of blood loss and he was transfused. A barium series was normal. Esophagoscopy and gastroscopy revealed only a small hiatal hernia without bleeding. His symptoms abated and the patient was finally discharged on January 15, 1960, from which date he has been well.

The features of this case conform very well with the diagnostic criteria described above. It is unfortunate that choledochotomy was not carried out at exploratory laparotomy, as the site of bleeding might well have been identified.

Case 2.—R.G., (No. 316959), a 13-year-old boy, was seen at a local hospital on September 18, 1960, and was immediately referred to The Hospital for Sick Children, Toronto. He had fallen on to a pavement from a height of eight feet, landing on his right chest and abdomen. His symptoms were right upper abdominal pain and right shoulder-tip pain, worse on deep inspiration. The patient was pale and perspiring; his pulse rate was 120/min. and his blood pressure 120/60 mm. Hg. Bruising was evident over the right lower rib cage anteriorly. Abdominal examination revealed diffuse mild tenderness and voluntary guarding, maximal in the right upper quadrant. Rebound tenderness was present in all quadrants and there was rectal tenderness. His hemoglobin was 13 g. %; his urine was clear, and the thoracic and abdominal roentgenograms were normal.

On admission the diagnosis was "closed hepatic injury of undetermined extent". Treatment consisted of nasogastric suction, intravenous fluids and sedation. Gradual improvement ensued and he was well on discharge 12 days after the accident.

The boy was readmitted four days after discharge with episodes of severe upper abdominal pain associated with grossly bloody stools. Tenderness in the right upper quadrant was present. Colic recurred along with hematemesis. Upper gastrointestinal roentgenography was normal and the gallbladder failed to concentrate any dye. One week after readmission mild jaundice was first noted. During the next week further attacks of pain and bleeding occurred requiring transfusions and frequent analgesics. On October 23, 1960, five weeks after the accident, a very severe attack of pain simulating biliary colic was observed, accompanied by massive hematemesis and bleeding per rectum.

Emergency laparotomy was carried out with a view to direct attack on the source of hemorrhage. A recently healed laceration over the dome of the right hepatic lobe was adherent to the diaphragm and was not disturbed. The gallbladder was tensely distended with blood clot, and possessed a diffusely edematous wall with several small necrotic areas over the fundus. To facilitate choledochotomy the gallbladder was then removed. On opening the common duct a cylindrical clot about 2 cm. to 3 cm. in length was removed from the right hepatic duct and bright red arterial blood escaped from the choledochotomy. This bleeding persisted and became alarming. Because of deterioration in the patient's general condition, the main hepatic artery was occluded by blunt pressure, immediately stopping the hemorrhage. Upon release of the pressure almost instantaneous resumption of profuse arterial hemorrhage was noted. A second occlusion of the artery was maintained for 10 minutes and was again unsuccessful in preventing further bleeding. At this point ligation of the right hepatic artery was de-

cided upon and quickly carried out, completely controlling the hemorrhage. No marked colour change was noted in the right hepatic lobe. The abdomen was closed after a T-tube had been inserted into the common bile duct. In the recovery room intravenous administration of penicillin and tetracycline was begun.

Postoperatively the T-tube drained some old blood for two days, after which the drainage consisted of clear bile. The patient ran an afebrile, uncomplicated course, taking his antibiotics orally by the third day and a full diet by the fourth. The T-tube was removed on the tenth day. The antibiotics were discontinued on the fourteenth day and the patient was discharged on the sixteenth postoperative day with his wounds well healed.

This boy remained well to the date of his most recent follow-up examination four months after the ligation of his right hepatic artery, at which time he was participating in all activities.

In Case 2 hemobilia was suspected before the exploration. Ligation of the right hepatic artery was performed because of rapid deterioration of the patient's condition. Postoperative antibiotics were used to prevent the clostridial hepatitis which might occur in a hypoxic liver. Penicillin and tetracycline were selected as the antibiotics of choice because of the known effectiveness of the former against most clostridia and the ability of the liver to concentrate the latter in bile.

Treatment

Many cases of hemobilia secondary to liver injury do not come to operation. In most of those who have been operated upon, choledochal irrigation and drainage through a T-tube has been sufficient. It is difficult to say what is really accomplished by this technique apart from evacuation of blood clot. Direct attack on the bleeding site, requiring a thoracoabdominal exposure, has been carried out in two reported cases, successfully in both instances.[2, 3] Hepatic lobectomy has also been suggested.

Ligation of the hepatic artery or one of its major branches has been used in the treatment of portal hypertension and ruptured aneurysm of the hepatic artery. As demonstrated by the second case, it may also be used in the treatment of traumatic hemobilia, provided adequate antibiotic coverage is maintained.

Discussion

In 1949, Markowitz, Rappaport and Scott[4] showed that the high mortality rate following hepatic artery ligation in dogs was due to clostridial proliferation in the hypoxic liver, with lethal gas gangrene as the outcome. Markowitz[5] wrote that the function of the hepatic artery was merely to discourage proliferation of anaerobes in the liver, which is constantly receiving them from the gut and that this function could also be provided by antibiotics. It was suggested that clostridia thrive in tiny infarcts resulting from the arterial occlusion and that if their growth were inhibited until the infarcted areas were phagocytosed, the drugs could then be discontinued. In mice, one injection of aqueous penicillin lasting four hours was sufficient for this purpose.[6] In man, antibiotic coverage for a two-week period is recommended. It is interesting to note that some of the dogs, whose hepatic arteries were tied and who received antibiotics, died from gangrene of the gallbladder and bile peritonitis. This was due to occlusion of the arterial supply to the gallbladder along with the right hepatic artery. For this reason cholecystectomy is recommended whenever the main or right hepatic artery is ligated.

It should be emphasized that hepatic artery ligation should be used only for those with continuing active massive hemorrhage.

Summary

The literature on traumatic hemobilia is reviewed and its diagnosis is discussed.

Two cases, including one with operative confirmation, are presented demonstrating this condition.

Ligation of the appropriate main hepatic arterial branch is shown to be safe and immediately effective in stopping massive traumatic hemobilia.

The importance of antibiotic support is discussed, as is the rationale for its use.

The need for cholecystectomy when the hepatic artery is ligated is mentioned.

Addendum

Since the submission of this manuscript two other references pertaining to traumatic hemobilia have been brought to our attention. Spector[10] described a case of traumatic hemobilia, in a 43-year-old man who had sustained a closed injury to his right abdomen, that finally required ligation of the right hepatic artery as a life-saving measure. This was done during the course of the patient's fourth operation. Recently Saliba *et al.*[11] reported a case of traumatic hemobilia in a 14-year-old boy as the result of a bullet wound. This was treated by direct approach to the bleeding site and transfixion of the area. The amount of liver destruction during this boy's illness was followed by serial transaminase studies.

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Résumé

Une hémorragie massive de l'arbre biliaire est une complication possible des traumatismes fermés du foie: Sandblom en 1848 décrivit le premier cas de ce genre et lui donna le nom "d'Hémobilie traumatique".

Cet accident est peu fréquent: on en trouve à peine une quinzaine de cas dans la littérature mondiale, et la plupart des manuels de chirurgie omettent d'en parler.

Le présent article donne l'histoire de deux nouveaux de ces cas.

Le premier est celui d'un garçon de huit ans, admis à l'hôpital à la suite d'un accident d'automobile. Il présentait des fractures multiples (tibia droit, péroné et clavicule) ainsi que des lésions abdominales internes se manifestant par des douleurs généralisées et un certain état de contracture de la paroi. Ces symptômes ayant spontanément régressé, l'enfant fut autorisé à quitter l'hôpital 12 jours plus tard. Il fallut cependant le réadmettre peu après pour une crise abdominale aiguë avec hématémèse et selles sanglantes. Une laparotomie exploratrice fut faite, qui ne montra aucune cause d'hémorragie. Des examens complémentaires pratiqués ultérieurement (radiographies à vide, transit baryté, œsophagoscopie et gastroscopie) ne révélèrent rien d'anormal; les symptômes disparurent d'eux-mêmes et à l'heure actuelle, le malade semble guéri. Dans ce cas, le diagnostic n'est fait que de façon rétrospective.

Le deuxième cas est celui d'un garçon de treize ans qui avait fait une chute sur un trottoir d'une hauteur de 8 pieds. Il souffrait d'une douleur violente dans l'hypochondre droit avec irradiation dans l'épaule; son faciès était pâle, son pouls battait à 120 par min. Le diagnostic d'entrée était: "contusion hépatique de gravité indéterminée". On institua un traitement conservateur: aspiration par sonde nasale, liquides intra-veineux et calmants. Cet épisode guérit bien, mais il en survint ultérieurement plusieurs autres, parfois compliqués de jaunisse. On pratiqua une laparotomie exploratrice et l'on découvrit ainsi une blessure du dôme hépatique et une hémorragie diffuse des voies biliaires: la vésicule était distendue par un gros caillot et fut enlevée. Ceci permit l'exploration de l'arbre biliaire, dont on put extraire un deuxième caillot cylindrique long d'environ un pouce. Une hémorragie survint alors, suffisamment abondante pour que l'on se résigne à ligaturer l'artère hépatique droite. L'abdomen fut refermé en laissant en place un drainage en T. Les suites opératoires furent bonnes et le malade est maintenant tout à fait guéri.

SPONTANEOUS RUPTURE OF THE ESOPHAGUS*

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SPONTANEOUS rupture of the esophagus has been recorded in medical literature for over 200 years,[5] but only two cases are known to have survived before 1947, when Barrett,[3] and almost simultaneously, Olsen and Clagett[21] reported successful surgical treatment of cases. Since then many reviews[1, 2, 9, 14, 16, 22, 23, 25] indicate that the condition is not as rare as was once believed, though the mortality rate is still over 30%.[2, 9, 16]

The diagnosis of spontaneous rupture of the esophagus is easy if the condition is considered. However, the characteristic symptoms and signs are sufficiently uncommon that the correct diagnosis may not be entertained during the important first few hours when surgical therapy is most effective. In none of the nine cases to be presented in this report was the condition suspected initially, and in four it was never considered till demonstrated at autopsy. The differential diagnosis includes perforated duodenal ulcer, bleeding duodenal ulcer, acute pancreatitis, myocardial infarction, spontaneous pneumothorax and pulmonary embolism. Of these potentially lethal emergencies, only spontaneous rupture of the esophagus requires urgent thoracotomy to save life. It is hoped that recapitulation of the clinical picture will lead to its early recognition, early operation and a fall in the still formidable mortality rate.

The term "spontaneous rupture of the esophagus" has been retained in the medical vocabulary in spite of seemingly sound arguments against its use.[6, 23] All cases of rupture of the apparently healthy esophagus not due to direct or indirect trauma are included under this term. Nine such cases, found in the records of the Toronto General Hospital, fall naturally into two groups — four patients (cerebral group) suffered esophageal rupture after intracranial hemorrhage; five others (emetic group) suffered rupture during a bout of violent vomiting.

REVIEW OF CASES

I. *Cerebral Group*

This group included two men and two women. Their ages ranged from 18 to 56 years. Three patients had intracranial bleeding after neurosurgical procedures (two after removal of a pituitary adenoma and one after removal of a temporal lobe astrocytoma). The fourth patient had massive intracranial hemorrhage as a complication of hypertension. No signs or symptoms except marked terminal hyperthermia were recorded, and in no case was the correct diagnosis made before death.

At autopsy three patients had suffered perforation of the esophagus on the left side, and one had perforated on both right and left sides. Perforations were multiple in three patients and single (left side) in one. In every case special note was made of the extreme friability of the esophagus and stomach. It is unlikely that surgical therapy could have been effective in this group.

II. *Emetic Group*

This group was made up of four men and one woman. Their ages ranged from 45 to 71 years. One patient was a chronic alcoholic and another had overindulged on the day of his rupture. Four of the five gave a definite history of duodenal ulcer; three of these had passed tarry stools during the previous week. Rupture occurred on the left side in three men, and on the right side in one man and in one woman.

History

All five patients gave a history of violent vomiting during which they suffered very severe substernal or epigastric pain. The pain radiated to the back and to the side of the rupture in three. Of the other two patients, one (with rupture on the right)

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Presented at the annual meeting of the Royal College of Physicians and Surgeons of Canada, Ottawa, January 1961.

complained of diffuse abdominal pain with bilateral shoulder-tip pain and the other (ruptured on the left) had bilateral lower chest pain radiating to the back. All noted intensification of pain by breathing, coughing or further efforts at vomiting. Aggravation of pain by breathing led to dyspnea in four. In every case it was immediately obvious to the patient that he was seriously ill and a physician was called within minutes. The initial diagnosis was perforated duodenal ulcer in four cases and myocardial infarction in one. Each patient was given a full dose of either morphine or meperidine (Demerol), but in no case was the pain relieved.

None of these patients experienced the sensation of something bursting in the chest, nor did any develop voice change or massive subcutaneous emphysema, though these symptoms are described in the literature.[2, 14, 24]

Physical Examination

All patients were in shock when they reached hospital. All were restless, three were slightly cyanosed, and most found greatest relief from their painful grunting respirations in the sitting position. One man refused to talk because the effort was painful.

Examination of the neck disclosed definite surgical emphysema in two patients, and jugular distension due to mediastinal venous obstruction in another.

Evidence of intrathoracic abnormality was found on physical examination in all patients, early in four and after 18 hours in the other. One man had a tension pneumothorax that required emergency decompression. Two other patients had definite signs of pleural effusion and the remaining two had pneumonitis. All signs were on the side of the lesion. In two patients a mediastinal "click" or "crunch", indicative of air in the mediastinum, was found.

In every case abdominal examination led to suspicion of acute intra-abdominal disease. All patients had ileus. In one, generalized abdominal rigidity was noted; in three patients rigidity was present but confined to the upper abdomen; only in one patient was rigidity not recorded. Every chart recorded some abdominal tenderness but most stated it was remarkably slight for the degree of abdominal rigidity present. No patient had loss of liver dullness.

Laboratory Examination

A chest radiograph, when it became available, was the most valuable laboratory aid. (Unfortunately it was not always ordered initially as most of these patients in the early stages were thought to have perforated ulcer). All patients had pneumonitis on the side of the lesion. Four patients had hydropneumothorax on the side of the lesion. In one of these, and in one who had only pneumonitis, air was visible on the radiograph in the mediastinum and in the subcutaneous tissue of the neck.

Absolute confirmation of the diagnosis was obtained twice with lipiodol esophagrams, which clearly demonstrated the site of rupture. Barium was not used because of its irritating properties.

Abdominal radiographs were obtained in the four patients thought to have a perforated duodenal ulcer. All showed mild ileus; none showed free air under the diaphragm. Free air under the diaphragm has never been reported in this condition.[1, 7, 22]

Chest aspiration was carried out in three patients. In each, a large quantity of fluid, recognized as gastric contents in two, was obtained. The test in which dye is given by mouth and recovered in the pleural aspirate is diagnostic, but was not used in this series.

Treatment

Initially all patients were treated for shock with plasma expanders followed by blood. All received massive doses of antibiotics. Two patients did not have a thoracotomy; of these one survived and one died. The first, a 49-year-old woman, survived a seven-hour period of hypotension, followed in convalescence by a multiloculated empyema which eventually responded to conservative measures. She later underwent partial gastrectomy for duodenal ulcer with pyloric obstruction. The second, a man aged 45, remained undiagnosed till autopsy. During the course of his

illness he was subjected to a fruitless laparotomy for suspected perforated duodenal ulcer.

Three patients underwent thoracotomy; two survived and one died. The two survivors, men aged 69 and 45 years, each had a left thoracotomy, suture of the rent in the esophagus and wide drainage of the mediastinum. In the first patient the vertical tear (lying just above the diaphragm on the left side posteriorly) was closed with a single layer of interrupted 00 chromic sutures. This man had an uncomplicated recovery. The second patient had a similarly placed tear sutured in two layers (inner running 00 chromic and outer interrupted silk). During the postoperative period he developed an esophageal fistula and pyopneumothorax, but these responded well to conservative measures.

The third man, aged 72, had a left thoracotomy for right-sided rupture. At operation it was not possible to find and suture the laceration in the esophagus, and although the mediastinum was drained widely, the patient died of mediastinitis and a loculated empyema medial to the right lung.

Discussion

The two factors which could lead to rupture of the esophagus are raised intraesophageal pressure and weakening of the esophageal wall. Clinical and laboratory evidence may be assembled to support either factor as being of primary importance,[6, 14, 16] but it is likely that in many cases both are operative.

Since most cases of spontaneous rupture of the esophagus have occurred during vomiting, parturition, weight lifting or straining at stool,[15, 16] there can be little doubt that raised intraesophageal pressure is an important cause of the condition. In the cadaver, with the organs *in situ*, it has been repeatedly demonstrated that the esophagus ruptures at about 5 lb. per square inch pressure.[14, 16] The rupture is single, 2 to 5 cm. long, and almost always occurs on the left side just above the diaphragm. With the esophagus removed from the body this site is also the first to rupture, for in this region there are weak areas in both the circular muscle, where vessels and nerves enter the esophagus, and in the longitudinal muscles, where they fan out to pass on to the stomach.[18] It is probable that in violent, uncoordinated vomiting, sufficient pressure may be produced in the normal esophagus to rupture its wall: uncoordinated spasm of the cricopharyngeus during maximal contraction of the abdominal wall and diaphragm would be particularly hazardous.[1, 18, 23]

Weakening of the esophageal wall, asymptomatic and unrecognizable because of the general inflammation after rupture, may be equally important in the causation of this lesion. None of the patients in the cerebral group was known to have vomited, though this may have occurred and not been recorded in these seriously ill people. Other patients are reported to have suffered rupture without vomiting and without cerebral disease.[24] Cushing[10] (in the fourth Balfour Lecture delivered at the University of Toronto in 1931) reemphasized the association of ulceration and perforation of the esophagus, stomach, and duodenum with intracranial lesions. This had been noted 90 years earlier by Rokitansky. Cushing pointed out, as we have noted in the cerebral group, that perforation most commonly followed craniotomies but could occur apart from this association. The mechanism of production of the extreme friability with ulceration and perforation of the upper gastrointestinal tract has never been clarified.[4, 12]

Brackney *et al.*[6] have clearly demonstrated in cats and dogs that esophageal rupture may be produced by the action of acid-peptic juice without any rise in intraesophageal pressure. In their animals there was a linear laceration on the left side just above the diaphragm (the usual site of clinical rupture). More recently Derrick, Harrison and Howard[11] have shown that the pressure required to cause esophageal rupture fell from about 5 lb. per square inch to slightly less than 2 lb. per square inch if the esophageal mucous membrane was removed. In the emetic group, four of the five patients had a known duodenal ulcer, and three had had tarry stools the week before their esophagus ruptured. Possibly these patients may have had a

mild acid-peptic weakening of their esophagus which predisposed it to rupture.

The onset of severe epigastric or substernal pain during vomiting, together with symptoms and signs suggesting both chest and abdominal disease, should alert one to the possibility of spontaneous rupture of the esophagus. Subcutaneous emphysema in the neck with such a history is diagnostic. A chest radiograph is then mandatory. Mediastinal air, demonstrable in the radiograph, can be caused only by spontaneous rupture of the esophagus, by rupture of the tracheobronchial tree, or by rupture of a tension pneumothorax into the mediastinum. A lipiodol esophagram will clarify the diagnosis and define the site of rupture. Not diagnostic but highly suggestive radiological signs are hydropneumothorax on the side of the lesion (the left side in 80%)[8, 9] and absence of free air under the diaphragm.

The urgency of early thoracotomy on the side of the rupture cannot be over-emphasized. The shocked patient will improve dramatically when gastric contents are removed from the pleural cavity. The esophageal laceration is usually found with ease. In the occasional difficult case, help may be obtained by asking the anesthetist to instil a small amount of methylene blue through a duodenal tube positioned just above the suspected point of rupture. After closure of the esophageal laceration the mediastinal pleura should be left widely open and the chest drained. The technique of closure does not seem to have influenced the occurrence of fistulae (about 30% are reported in the literature),[9] but fortunately these fistulae almost always respond to conservative therapy.

Cases seen late, certainly after 48 hours, are best treated conservatively with multiple thoracenteses or with tube drainage of the chest if indicated.

Summary

Spontaneous rupture of the esophagus is a highly lethal condition that is amenable to surgical therapy.

Of the nine cases seen at the Toronto General Hospital, four fall into a "cerebral" group and five into an "emetic" group.

In the cerebral group multiple ruptures of a friable esophagus occurred as a terminal event in a patient moribund after intracranial hemorrhage. Little could have been done for these patients, all of whom died.

In the emetic group a single laceration immediately above the diaphragm occurred during a bout of violent vomiting. Two were treated without operation and one survived. Three were operated upon; the two in whom it was possible to suture the rent in the esophagus survived; the third, in whom mediastinal drainage only was carried out, died.

Early recognition and suture of the rupture offers hope of reducing the mortality of this still lethal condition.

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RÉSUMÉ

Des cas de rupture spontanée de l'œsophage ont été publiés dans la littérature chirurgicale depuis deux cents ans. De tout cela, il ressort que cet accident n'est pas aussi rare qu'on le pense et que la mortalité est aux alentours de 30%.

Le diagnostic en est facile, à condition d'y penser. Le présent rapport se réfère à neuf cas de ce genre, parmi lesquels quatre n'avaient été diagnostiqués qu'à la salle d'autopsie. Le diagnostic différentiel est à faire avec l'ulcère duodénal perforé, la pancréatite aiguë, l'infarctus du myocarde, le pneumothorax spontané et l'embolie pulmonaire. Quatre des malades présentaient une rupture œsophagienne consécutive à une hémorragie intra-crânienne, et les cinq autres à la suite de violents efforts de vomissement (groupe cérébral et groupe émétique).

Dans le groupe cérébral il y avait deux hommes et deux femmes dont les âges variaient entre 18 et 56 ans; l'hémorragie intra-crânienne avait été causée par de l'hypertension (1 cas) ou des interventions neurochirurgicales (3 cas). Le diagnostic n'avait pas pu être posé; à l'autopsie, on note toujours une extrême friabilité de la paroi œsophagienne, de sorte qu'il semble que tout essai de réparation chirurgicale aurait été voué à l'échec.

Le groupe émétique comprenait quatre hommes et une femme âgés de 45 à 71 ans. Tous avaient eu, après des vomissements très violents, des douleurs rétrosternales subites et atroces, exagérées par la respiration, la toux et les nausées. A l'examen physique, on trouva les signes suivants: état de choc avec parfois cyanose; distension de la jugulaire ou emphysème sous-cutané dans la région cervicale; état d'iléus abdominal; contracture de la paroi abdominale de degré variable.

Parmi les tests de laboratoire, le plus utile est la radiographie simple du thorax qui montre toujours une image pneumonique du côté de la lésion, avec parfois une image hydro-aérique pleurale. Une injection de lipiodol dans l'œsophage permet de situer exactement la blessure.

Le traitment consistera à réduire les symptômes de choc par des transfusions de plasma et de sang. Du point de vue chirurgical, la seule thérapeutique valable est la thoracotomie, aussi précoce que possible, qui permettra de découvrir la lésion et de la réparer. Il est important de savoir que le simple fait de l'épanchement intra-pleural de liquide gastrique contribue considérablement à la gravité du choc; l'évacuation de cet épanchement améliore toujours de façon spectaculaire l'état du patient.

PREFERENCES IN SUTURE MATERIAL

"Nylon was one of the earliest synthetic surgical suture materials, and observations of its behaviour in the various tissues of the body have been required to determine possible uses in surgery. Some surgeons after experiencing difficulties with nylon have banished it decisively, returning to 'good old catgut' as whole-heartedly as Halsted in his time, after catgut troubles, uttered his peremptory, 'Away with the catgut', and ushered in a silken era.

"But sutures, like man himself, are not all things to all men at all times in all places, though some try to make them so. Some go so far as to shun the use of any 'unabsorbable' suture, and some will never use catgut. Similar disagreement about the composition of 'strings' and 'lines' is said to be found amongst those interested in rackets and rods.

"Most would agree that, on the whole, nylon makes a satisfactory skin suture and catgut does not. Most would agree that nylon, along with other unabsorbable sutures, is unsuitable for use on the mucosal aspects of the urinary tract owing to calculus formation about such sutures, whereas catgut is the favourite when a suture must be left exposed to the lumen of urinary or biliary passages."

MOLONEY, G. E.: The effect of human tissues on the tensile strength of implanted nylon sutures, *Brit. J. Surg.*, **68**: 528, 1961.

L'INFILTRATION PERIDURALE CONTINUE DANS LES FRACTURES MULTIPLES DE COTES*

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EN THÉRAPEUTIQUE anesthésique, l'infiltration péridurale continue est utilisée surtout pour faire céder un spasme vasculaire, améliorer la circulation sanguine dans les membres inférieurs des artérioscléreux, soulager la douleur causée par une thrombose, ou comme traitement de la pancréatite aiguë ou sub-aiguë.

Depuis mars 1960, nous avons eu l'occasion de faire des infiltrations péridurales continues dans des cas de fractures multiples de côtes.

Le but en est de soulager la patient, et d'augmenter les mouvements de ventilation. Ceci permet de lutter contre l'atélectasie qui se produit souvent chez ces blessés.

Pour l'introduction du cathéter, si possible entre L1-L2, nous préférons la position latérale, l'emploi de l'aiguille Tuohy N° 16, et un cathéter urétéral N° 3 en nylon à pointe mousse à ouverture bilatérale, opaque aux rayons X.

Comme substance analgésique, la Pontocaïne 0.15% est idéale parce que nous réussissons souvent à obtenir un blocage des fibres sensitives d'une durée de trois heures et plus.

La première fois, une injection de 15 à 30 ml. est nécessaire pour soulager la douleur. Par la suite, 10, 15 ou 20 ml. toutes les six ou huit heures pendant quatre à huit jours suffisent. Après l'infiltration péridurale, nous injectons 5 à 15 mg. de Wyamine i.m. pour prévenir la chute de tension artérielle possible due à la paralysie des fibres sympathiques touchées.

Pour illustrer notre exposé, voici un résumé des dossiers de six patients que nous avons eu à traiter depuis quelques mois.

1er Cas.—S.F., âgé de 47 ans, souffre de contusions du thorax et de l'abdomen, ayant été coincé entre deux camions le 3 mars 1960. Hospitalisé le 11 mars 1960, le patient est épuisé, anxieux, ne peut dormir, refuse de remuer, reste assis. Sa respiration est superficielle à 24 par minute. Une radiographie montre des fractures des 3-4-5-6-7-8-9èmes côtes droites et de la 9ème côte gauche, ainsi que de l'atélectasie pulmonaire à droite. Température normale. Trois heures après son arrivée, nous installons une sonde pour infiltration péridurale continue et nous injectons 35 ml. de Pontocaïne à 0.15%. L'anesthésie au toucher va de D2 à D10. Les fibres motrices ne sont pas touchées. L'analgésie apparait en moins de cinq minutes et dure trois à quatre heures.

Par la suite, nous avons injecté 20 ml. toutes les huit heures pendant trois jours, et 15 ml. pendant quatre jours. Le patient peut effectuer ses exercices respiratoires et dormir. L'atélectasie régresse.

La sonde qui a servi à la péridurale est enlevée le 18 mars, sept jours après son installation. Le patient continue son traitement sans analgésique.

2ème Cas.—Mme. A.S., âgée de 46 ans, commence à ressentir des douleurs thoraciques cinq heures après un brusque coup de frein qui l'a projetée contre le tableau de bord d'une automobile. Une injection de 1/32 de grain de Dilaudid toutes les quatre heures ne la soulage pas.

Elle est hospitalisée le 26 mars 1960; la radiographie montre des fractures des 5-6-7-8-9èmes côtes gauches, avec atélectasie du même côté.

On installe une sonde pour l'infiltration péridurale continue, et on injecte 35 ml. de Pontocaïne. L'analgésie va de D4 jusqu'aux jambes. Par la suite, il faut de 20 à 25 ml. toutes les huit heures pour apporter un soulagement et permettre à la patiente de tousser et de maintenir libres ses voies respiratoires.

Trois jours plus tard, on cesse l'infiltration péridurale parce que la patiente qui avait déjà été traitée pour gastrite éthylique, fait du delirium tremens. On néglige alors les fractures de côtes. La malade est traitée avec succès pour ce nouvel épisode.

3ème Cas.—H.C. est adressé à l'hôpital le 11 août 1960, huit jours après un accident d'auto. Il recevait du Pantopon toutes les deux heures, avait une respiration difficile et stertoreuse à

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24 par minute. Sa température était à 102°F., son pouls à 125/min. Son médecin le référait pour hernie diaphragmatique post-traumatique.

Pour diminuer la douleur costale, et surtout pour lui permettre de mieux respirer et de vider ses bronches des sécrétions, le Dr A. J. lui fit immédiatement une infiltration péridurale continue.

Du 11 au 15 août, le patient reçoit alternativement, toutes les quatre heures, 15 ml. de Pontocaïne à 0.15% et 100 mg. de Démérol. Le patient a des nausées et des vomissements. Sa respiration s'est améliorée.

Le 15 août, le malade a subi une thoracotomie pour la réparation d'une brèche traumatique longue de quatre pouces dans le diaphragme.

Au cours de la première journée post-opératoire, il a reçu alternativement, toutes les quatre heures, 100 mg. de Démérol et 15 ml. de Pontocaïne à 0.15%.

La seconde journée, il a reçu trois injections de Pontocaïne et deux injections de Démérol. Le patient a passé une bonne nuit.

Du 18 au 22 août, le patient n'a reçu que trois injections de Pontocaïne par jour. Il poursuit ses exercices respiratoires et circule.

Le patient quitte l'hôpital le 25 août, en très bon état.

4ème Cas.—L.D., âgé de 39 ans, débardeur, est hospitalisé à la suite d'un accident survenu le 27 septembre 1960. Une radiographie montre une fracture avec décalage sur l'axe axillaire postérieur des 3-4-5-6-7èmes côtes gauches, et une atélectasie du même côté. Le patient expectore du sang. A l'inspiration le patient souffre d'une douleur atroce; 75 mg. de Démérol le soulage pour une heure. Il refuse les aérosols à l'alevaire et les exercices respiratoires.

Le 29 septembre 1960, le Dr F. P. installe une sonde pour infiltration péridurale continue, et injecte au début, 20 ml. de Pontocaïne à 0.15%. Par la suite, il fut injecté 10 ml., toutes les six heures pendant les deux premiers jours, puis toutes les huit heures jusqu'au 5 octobre. Durant ces six jours, le patient circule, fait ses exercices respiratoires et se sent très bien.

5ème Cas.—E.L., âgé de 60 ans, hôtelier, fait une chute sur un bureau. Une radiographie faite le 15 octobre 1960, montre des fractures avec décalage des 7-8-9èmes côtes gauches et un pneumothorax du même côté. Le malade présente une suffusion hémorragique à droite et souffre beaucoup.

Le 15 octobre, on installe une sonde à infiltration péridurale continue, et on injecte 10 ml. de Pontocaïne à 0.15%, toutes les deux heures. Chaque injection lui permet de tousser et d'expectorer.

On remarque les 16 et 17 octobre, que le malade a une respiration à 30/min., alors que son pouls reste à 72/min. et que sa température est à 97.4°F. La tachypnée nous fait penser que l'atélectasie s'installe. Le pansement est changé pour permettre une respiration plus ample. Le 18, tout rentre dans l'ordre, et nous donnons de la Pontocaïne à 0.15% toutes les six heures, jusqu'au 21 octobre. Le patient quitte l'hôpital très heureux; il attribue sa guérison à "son tube dans le dos".

6ème Cas.—J.T., âgé de 48 ans, bûcheron, reçoit un arbre sur le dos. Une radiographie faite le 20 octobre 1960 montre une fracture des 6-7-8-et 10èmes côtes gauches, et un écrasement de la 11ème vertèbre dorsale. Sa respiration à 26/min. est superficielle.

Le 21 octobre, le Dr A. Mc. lui installe une sonde à infiltration péridurale continue et injecte 15 ml. de Carbocaïne à 1% et 10 à 20 ml. de Pontocaïne à 0.15% toutes les quatre à six heures, ceci jusqu'au 25 octobre. Le soulagement est bon.

Sa convalescence se poursuit sans que d'autre analgésique soit nécessaire.

Discussion

L'infiltration péridurale continue a un avantage sur l'infiltration para-vertébrale, ou l'injection dans les foyers de fractures. Celle-ci doit être répétée trop souvent pour arriver à un résultat semblable.

Une injection de 30 ml. de Pontocaïne à 0.15% est souvent nécessaire pour soulager la douleur lors de la première injection. Pour les injections suivantes, 10 à 20 ml. de Pontocaïne dans le cathéter suffisent à faire disparaître toute douleur, ce qui représente une dose beaucoup inférieure à celle utilisée dans les cas d'infiltrations multiples.

Comme prophylaxie contre la chute de la tension artérielle, on donne une dose d'un médicament vaso-constricteur proportionnelle à la quantité de Pontocaïne injectée.

A la suite de ces expériences, nous croyons, qu'à l'avenir, il faut penser à l'infiltration péridurale continue pour traiter les fractures multiples de côtes. Elle permet

de soulager le patient, de prévenir l'atélectasie et ses conséquences et de diminuer la dose d'opiacés.

En plus des contre-indications usuelles de l'infiltration péridurale, il y a le danger de masquer les symptômes abdominaux.

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SUMMARY

In this article the authors present the case histories of six patients who suffered multiple injuries, including rib fractures with or without pneumothorax, and one case of diaphragmatic hernia. In such circumstances, the usual surgical treatment may be supplemented by a continuous peridural infiltration of the posterior nerve roots. This technique is relatively simple and harmless and has many advantages, as it affords the patient complete relief from pain and permits him to breathe normally. In order to obtain the same analgesia with ordinary sedatives or tranquillizers, very large doses would have to be used. In practice the authors use a 0.15% solution of Pontocaine, which they inject in quantities of 10 to 20 ml. every four hours during the first few days after the injury.

CHIRURGIE A COEUR OUVERT ET ARRETE SOUS HYPOTHERMIE PROFONDE, DOUBLE CIRCULATION EXTRACORPORELLE ET AUTO-OXYGENATION*

Les méthodes permettant la réalisation d'interventions intracardiaques ne cessent d'évoluer. Après une croissance rapide, l'hypothermie modérée d'une part et la circulation extracorporelle avec cœur-poumon artificiel d'autre part, sont arrivés à maturité. Chacune connaît les limites de ses possibilités, ses avantages et ses inconvénients.

Simplicité pour la première, mais durée d'action très limitée; complexité pour la seconde (appareillage, quantité de sang donneur) mais possibilités étendues malgré les effets hématologiques et métaboliques des longues perfusions et la gène apportée dans certains cas par un retour sanguin intracardiaque anormalement abondant.

L'association récente des deux méthodes, constitue la dernière solution apportée au problème de l'assèchement cardiaque.

L'adjonction à l'hypothermie d'une assistance circulatoire artificielle a réduit à néant la menace de fibrillation ventriculaire, seul obstacle au franchissement des 28 degrés.

A l'heure actuelle, l'hypothermie profonde est le seul moyen de réduire le métabolisme au point que la circulation puisse être suspendue pour une période prolongée. Le refroidissement interne par circulation extracorporelle et à l'aide d'un échangeur de température, est le seul moyen de lui assurer l'efficacité et la sécurité nécessaire.

L'atteinte des basses températures, offrant des conditions idéales pour la chirurgie à cœur ouvert, peut être obtenue par deux méthodes: soit l'association d'un cœur-poumon artificiel et d'un échangeur de température; soit l'utilisation de deux circulations extracorporelles, l'une systémique avec échangeur de température, l'autre pulmonaire assurant une auto-oxygénation.

La première méthode, étudiée expérimentalement dès 1952 par Juvenelle, Gollan, Pierce, est utilisée depuis peu chez l'homme aussi bien aux Etats-Unis (Sealy, Longmire, Gerbode) qu'en France, où Dubost pouvait récemment rapporter les résultats de ses 36 premiers cas.

C'est vers la seconde méthode, mise au point et utilisée par Drew, Keen et Benazon, du Westminster Hospital de Londres, que nous nous sommes orientés dès le début de 1959. Après une expérimentation qui nous a confirmé les possibilités de cette méthode, nous en avons entrepris l'application humaine pour les lésions justiciables d'une intervention à cœur ouvert et en Janvier 1960 nous présentions à la Société Française de Cardiologie deux observations de Communication interventriculaire fermées avec succès à des températures de 10 et 11°.

*NÈGRE, E. *et al.*: *J. Chir.*, **80**: 178, 1960.

EXCISION OF THE CARPAL SCAPHOID FOR UNUNITED FRACTURES

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THE MERITS of excision of the carpal scaphoid have been debated for some time. Opinions vary in regard to the value of total as compared with partial excision. Dwyer[3] in 1949 reported generally good results from total excision in a group of 19 cases. He felt that in cases with partial excision the operation was difficult and impingement of the distal scaphoid against the radius was inevitable. Dwyer's series of patients with total excision included three very good, nine good, four fair and three bad results. Davidson and Horwitz[2] in 1938 also favoured total excision, reporting the results in their entire series of seven cases in 1938 as either good or excellent. The result in their single case of partial excision was not considered good. Obletz and Halbstein[4] favoured partial excision and reported four cases improved out of four so treated. Their series of three cases of complete excision included only one who was improved.

CLINICAL MATERIAL

This series includes 17 patients, of whom 12 had total excisions, three proximal pole excisions and two distal pole excisions. All patients were examined by the author and subsequently radiographed. The radiological examination included: (a) a lateral view in full dorsiflexion; (b) a lateral view in full palmar flexion; (c) an anteroposterior view in full radial deviation, and (d) an anteroposterior view in full ulnar deviation.

All of the patients were men. The right wrist was involved in nine instances and the left in eight. Fifteen patients were between 20 and 40 years of age; the remaining two were between 50 and 60 years. Twelve of the patients were operated upon within 10 years following the original fracture and five after a longer period. Non-union or delayed union was present in all the cases.

Various surgeons performed the operations. Excision of the radial styloid process was carried out in two of the proximal pole excisions.

POSTOPERATIVE ASSESSMENT OF PATIENTS WITH TOTAL SCAPHOID EXCISION

SYMPTOMS—(a) *Pain.*—All of the patients had pain in the wrist with activity. Four had only minor aching, five had moderate pain and three had sufficient pain to warrant a wrist arthrodesis. (b) *Ability to work.*—Four patients had to change to lighter work and five remained at similar occupations. This excluded those with arthrodesis of the wrist.

SIGNS (excluding patients with wrist arthrodeses)—(a) *Heat, fluid and thickening.*—Two patients had increased local heat at the time of examination. Thickening of the periarticular soft tissues was present in seven instances. (b) *Range of movement.*—The average ranges were dorsiflexion 37°, palmar flexion 41°, radial deviation 24°, ulnar deviation 25°. (c) *Crepitus.*—All showed fairly marked crepitus.

RADIOGRAPHIC OBSERVATIONS—(a) *Arthritic changes.*—Narrowing of the lunatocapitate articulation was present in eight instances. In one patient (Figs. 1 and 2), on whom the author operated recently for arthrodesis of the wrist, the presence of this change was noted in a relatively short time following scaphoid excision. Erosion of the articular cartilage and eburnation of the underlying bone was clearly seen. Radio-scaphoid joint narrowing was common preoperatively. In three instances the tip of the radial styloid was quite sharpened. No other radio-carpal or intercarpal narrowing was apparent either preoperatively or postoperatively. (b) *Carpal bone shift.*—Two patients who had an early arthrodesis of the wrist did not exhibit any carpal bone shift, nor was such a finding noted in one other patient not subjected to arthrodesis. This might have been because a dorsal prominence of the lunate

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Fig. 1.—Preoperative condition with nonunion of the scaphoid and apparent avascularity of the proximal pole.

prevented a dorsal shift of the capitate. In the remaining nine instances a shift was observed (Fig. 3). The capitate moved dorsally and radially on the lunate. The dorsal shift was marked in five, mild in one and absent in four patients. Radial shift was marked in five, mild in four and absent in one instance (Fig. 4). (c) *Cystic formation.*—This change was present in the capitate in five patients. No other bones were affected. (d) *Bone regeneration.*—This was marked in two, moderate in two, mild in four and absent in two instances.

Postoperative Assessment of Patients with Partial Scaphoid Excision

Symptoms—(a) *Pain.*—Intermittent minor aching on activity was noted by all patients. Pain was marked in one instance (Fig. 5), until secondary excision of the radial styloid process was performed, following which this patient's pain was minimal. (b) *Ability to work.*—All patients continued in the same occupation or in jobs that were equally arduous.

Signs—(a) *Heat, fluid and thickening.*—One patient exhibited minimal soft tissue





Fig. 2.—One year following excision of the carpal scaphoid (same case as in Fig. 1). There is narrowing of the lunato-capitate articulation and subluxation of the capitate dorsally on the lunate.





Fig. 3.—Eight years after excision of the carpal scaphoid. There is marked shift of the capitate dorsally and radially on the lunate.





Fig. 4.—Ten years after excision of the carpal scaphoid. There is absence of significant dorsal and radial shift of the capitate on the lunate. Note the large dorsal prominence of the lunate in the lateral radiograph.





Fig. 5.—Five years after proximal pole excision followed by radial styloid process excision. No shift of the capitate on the lunate.





Fig. 6.—Eight years after distal pole excision. No shift of the capitate on the lunate.

thickening. The others showed no evidence of heat, fluid or thickening. (b) *Range of Movement.*—The range of movement exceeded that of the patients in the total excision group. The average range was dorsiflexion 60°, palmar flexion 40°, radial deviation 23°, ulnar deviation 25°. No significant difference between the proximal pole excision group and the distal pole excision group was observed. (c) *Crepitus.*—Three patients had mild crepitus. (d) *Radiographic observations.*—There was no evidence of lunato-capitate joint narrowing, shift of the capitate or cystic formation in the carpal bones (Figs. 5 and 6).

Results of Total Scaphoid Excision

The results as shown in Table I were assessed on the basis of (a) pain; (b) range of movement; (c) radiographic changes, and (d) the need for change of occupation. *Excellent* (2 patients): In those patients whose result was classed as excellent there was no significant pain; the range of antero-posterior movement was 80° or more; the range of lateral movement was 50° or more; radiographic changes were absent or minimal, and there had been no change of occupation. *Good* (1 patient): This patient was so classified after a 22-year follow-up. Pain was minimal on heavy work. The range of movement was excellent. There was a mild radial shift of the capitate on the lunate. *Fair* (4 patients): These patients all had mild to moderate pain on heavy activity. In three the range of movement was excellent. A shift of the capitate dorsally or radially on the lunate was present in all instances. Narrowing of the lunato-capitate joint was present to a mild or moderate degree in all. *Poor* (5 patients): Three patients with severe pain were subjected to wrist arthrodesis. Two of these were elderly men who had long-standing delayed union or non-union of the carpal scaphoid. Arthrodesis was performed at an early date following excision. No significant carpal shift had occurred before operation. In the remaining patient with wrist arthrodesis, within the period of one year, fairly extensive changes resulted at the lunato-capitate joint with shifting of the capitate, narrowing of the joint and severe wrist pain. The remaining two patients with poor results had moderate pain on exertion. The range of movement was below an acceptable range and radio-

TABLE I.—Total Excision

Results	*Number of cases*
Excellent	2 (17%)
Good	1 (8%)
Fair	4 (33%)
Poor	5 (42%)

TABLE II.—Length of Follow-Up after Operation

Years	Results: Excellent	Good	Fair	Poor
0 - 2				
2 - 4			1	
4 - 6	1		1	
6 - 8	1			
8 - 10			1	
Over 10		1	1	2*
Total	2	1	4	2*

*Exclusive of cases with wrist arthrodesis.

logical evidence of carpal shift was present. However, both patients had declined arthrodesis in favour of preserving their mobility.

There was no apparent relationship of poor results to the length of follow-up period after operation (Table II).

There appeared to be an increased incidence of inferior results in patients who were operated upon 10 years or more following the initial fracture (Table III).

TABLE III.—Years Between Initial Fracture and Excision

Results	Years: 0 - 5	5 - 10	10 - 20
Excellent		2	
Good	1		
Fair	3		1
Poor		2	3
Total	4	4	4

Results of Partial Scaphoid Excision

These results were assessed by criteria similar to those described in the case of patients with total excision (Table IV).

TABLE IV.—Partial Excision

Results	Number of cases
Excellent	4 (80%)
Good	1 (20%)

Excellent (4 patients): One patient was elevated to this group after the secondary operation of radial styloid process excision. *Good* (1 patient): In this instance the distal pole of the scaphoid was excised. There was some limitation of wrist movement.

Discussion

Excluding the two patients who had early wrist arthrodesis, it was noted in nine of the remaining 10 instances of total scaphoid excision that a shift of the capitate on the lunate occurred. This shift appeared to bear a relationship to the production of pain. This relationship was not a complete one, however, as in some patients with severe shift, pain was only minimal. Narrowing of the lunato-capitate joint was noted in all patients except the two with excellent results and the two cases of early wrist arthrodesis, and this appeared to be an unfavourable observation.

In the partial scaphoid excision group, carpal shift or narrowing at the lunato-capitate joint was not seen. In two cases, excision of the radial styloid process as recommended by Barnard and Stubbins[1] in 1948 had been performed. This secondary operation improved the result in one of these patients.

Conclusions

Twelve patients with total scaphoid excision and five with partial scaphoid excision have been reviewed.

The overall results of total excision are not favourable and do not compare with those of partial excision.

A shift of the capitate on the lunate and a narrowing of the capitate-lunate articulation were unfavourable signs in most instances.

The results of total scaphoid excision in the elderly were poor.

The results of total scaphoid excision in patients with ununited fractures of long duration were generally inferior.

Acknowledgments

The author wishes to acknowledge the advice and encouragement of Dr. F. P. Patterson, Director, and Dr. K. S. Morton, Assistant Director, of the Trauma Research Unit. He wishes also to thank Miss Kay Hoskins for preparing the illustrations.

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RÉSUMÉ

La valeur thérapeutique de l'excision du scaphoïde carpien a été passablement discutée.

Le présent article se propose de jeter un peu de lumière sur cette question en présentant une étude d'ensemble de 17 cas de ce genre. Les malades étaient tous des hommes dont les âges variaient entre 20 et 40 ans. Douze d'entre eux avaient été opérés dans les dix ans suivant immédiatement la fracture et les cinq autres plus tardivement. Les excisions qui furent pratiquées étaient totales (12 cas) et partielles (5 cas).

Dans l'ensemble, les résultats de l'excision totale sont mauvais ou médiocres et ne se comparent pas à ceux de l'excision partielle. Un déplacement du grand os par rapport au semi-lunaire, et le rétrécissement radiologique de l'articulation entre ces deux os sont des signes de mauvais pronostic. Les résultats de l'excision totale furent toujours mauvais chez les patients les plus âgés, de même que chez ceux où il y avait eu fracture non-consolidée longtemps auparavant.

CORTICOSTEROID-INDUCED ADRENAL SUPPRESSION

"In order to select a therapeutic program that will allow a patient with corticosteroid-induced adrenal suppression to withstand a surgical operation, it is essential to be cognizant of the physiology involved in the development of an adrenal crisis postoperatively. Swingle and colleagues have demonstrated that the greatly increased susceptibility of adrenalectomized dogs to stress induced by a minor operative procedure is apparently due to severe dehydration of the extracellular compartment owing to the passage of water into cells. Plasma volume and blood pressure decline, eventuating in circulatory collapse. The fluid so shifted is held immobile within the cells, presumably by osmotic forces which the animal is powerless to overcome in the absence of glucocorticoids despite the desperate need to rehydrate the extracellular space and maintain circulation. When a glucocorticoid is injected, the adrenalectomized animal is able to redistribute intracelluar fluid and increase the plasma volume, thus restoring circulatory efficiency. It is important to bear in mind that potent mineralocorticoids, such as aldosterone or desoxycorticosterone, exert only a minimal effect on the distribution of intracelluar fluid and are incapable of relieving the severe symptoms of adrenal insufficiency, either in dogs or man.

"It seems appropriate to consider the relationship between the plasma level of a glucocorticoid and its biologic activity. In discussing this relationship, it must be remembered that the recently developed synthetic corticosteroids are more effective physiologically on a weight basis than cortisol, a naturally occurring adrenal hormone. Two possibilities exist as explanations for this quantitative difference in potency between the synthetic corticosteroids and cortisol. First, the synthetic cortisol analogues, of which prednisolone is representative, may be metabolized at a slower rate by the liver than is cortisol. Indeed, prednisolone has been shown to have a biologic half-life of 191 minutes in human plasma after its infusion while the corresponding half-life of cortisol is only 114 minutes. This observation indicates that a given dose of prednisolone should remain in the circulating blood longer before it is completely metabolized than an identical amount of cortisol. That a decreased clearance rate of prednisolone from the blood is solely responsible for its enhanced biologic activity seems unlikely. A second explanation is suggested by the work of Glenn, who injected 1.0 mg. of prednisolone and 1.0 mg. of cortisol (in 0.1 ml. of ethanol) into the tail veins of fasted adrenalectomized rats and found that the liver glycogen deposition response to prednisolone lasted for 32 hours, whereas cortisol exerted a similar effect for only 8 hours. The results of this experiment suggest that predisolone may persist for longer periods in tissue fluids than does cortisol. Thus, after prednisolone has completely disappeared from the blood, it still may be capable of influencing metabolic processes operating at a cellular level."

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EXPERIENCE IN THE SURGICAL MANAGEMENT OF DUODENAL AND GASTRIC ULCERS*

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A CONSIDERABLE interest in the surgical management of peptic ulcer patients, coupled with the responsibility for the care of certain problem cases resulting from faulty and inadequate treatment of penetrating duodenal ulcers in particular, has led the writer to carry out an extensive review of a personal series extending over the last 20 years (1940-1959). An attempt has been made to include every patient treated surgically for duodenal or gastric ulceration, except those patients whose operation was directed solely to the closure of a perforation.

CLASSIFICATION AND SEX DISTRIBUTION

Patients have been classified according to whether the ulceration was duodenal or gastric. Both types of ulcer were present in a small group. The duodenal cases have been divided into those with or without penetration. Those with anastomotic ulcers subsequent to previous gastric procedures have constituted a group by themselves. This classification, together with the breakdown according to sex distribution and age incidence, is depicted in Tables I and II, covering the entire series of 326 cases.

TABLE I.—SEX DISTRIBUTION

Type of ulcer	*Male*	*Female*	*Total*
Duodenal ulcer penetrating	148	24	172
Duodenal ulcer non-penetrating	43	13	56
Duodenal ulcer and gastric ulcer co-existing	21	4	25
Gastric ulcer	35	24	59
Anastomotic ulcer	12	2	14

The overall ratio of males to females is 4:1, with some variation in the different types. This is in accord with the established fact that parietal cell counts are higher in the male than in the female. Almost all ages were represented, with preponderance in the fourth and fifth decades. The youngest patient having a definitive operation for a penetrating duodenal ulcer was 17. His ulcer had perforated three years earlier, and this was followed by repeated serious hemorrhages and resistance to treatment. Ten years later, he is married, has a family, and enjoys perfect health. The oldest patient was 87 years of age and suffered exsanguinating bleeding from a penetrating gastric ulcer. The relative frequency with which ulceration in both the stomach and duodenum was found at the same time seemed a little surprising. Study of many records also brought out conflicting radiological findings in a number of instances: a duodenal ulcer being reported unequivocally one year, a definite gastric lesion the next year, and so on. Clear-cut separate ulcers were noted in most of those classified under the combined heading. However, there were a very few situations in which pyloric ulceration seemed to extend both ways so much that no other heading seemed appropriate.

DIAGNOSIS AND ASSESSMENT

The majority of the patients had long clinical histories, more or less typical for the particular ulceration found, symptoms becoming more persistent and resistant to therapy with the passage of years. The incidence of complications was high. Usually, there had been repeated hospitalizations, and programs of medical management had been followed reasonably faithfully for a matter of years. In the penetrating duodenal ulcer cases it was customary to find that the bouts of periodic pain had given way to severe pain, boring

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*Presented in summary at the Sectional Meeting, Royal College of Physicians and Surgeons of Canada, London, Ont., November 15, 1960.

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TABLE II.—Age Incidence

	Under 30	*30 to 39*	*40 to 49*	*50 to 59*	*60 to 69*	*70 to 79*	*Over 80*	*Total*
Penetrating duodenal ulcer	10	21	47	47	32	15	—	172
Non-penetrating duodenal ulcer	—	7	22	14	8	2	3	56
Duodenal and gastric ulcers	2	2	5	12	2	2	—	25
Gastric ulcer	2	3	12	23	14	4	1	59
Anastomotic ulcer (14 original—16 acquired)	1	4	11	6	5	3	—	30

through from the epigastrium to the back, present much of the time, not responsive to food, alkalis or drugs, and causing great disability.

Serial roentgenological examinations were available in the vast majority, affording much assistance, but not always telling the whole story. Laboratory studies from the records of the various hospitalizations proved helpful guides. Preoperative acid studies were augmented in many cases by measurement of night secretion (volume and acidity), and all accepted aids were employed whenever this was deemed advisable.

Incidence of Ulcer Complications

The ulcer complications in the series as a whole are summarized in Table III, while

TABLE III.—Incidence of Ulcer Complications in Series

Complications	*Number of cases*	*%*
Perforation	40	12.2
Hemorrhage	120	36.8
Stenosis	75	23.0
Stomal ulceration	14	4.3

the incidence of each of the common complications in relation to the type of ulcer is indicated in Table IV.

These figures show that for the penetrating duodenal ulcer each complication has an incidence substantially greater than the average for the series. They fail to reveal an additional fact of importance, namely, that among the duodenal cases shown in Table IV there were several examples of multiple perforations (one patient had four, two had three and four had two). Many of those listed under hemorrhage bled badly and repeatedly. A fair percentage of these patients were admitted to hospital in the stage of acute hemorrhage, and 13 had an emergency gastric resection because of persistent exsanguinating bleeding (four had penetrating duodenal ulcers, one had duodenal and gastric ulcers, seven had gastric lesions, and one developed gastric and stomal ulcerations after a Billroth I resection). Many patients had more than a single complication at one time or another.

Indications for Operation and Choice of Procedure

By and large, the operative indications were those generally accepted today – complications and intractability (commonly related to penetration). Special considerations arose in several cases. These included family and past history, response to therapy, ability and willingness to co-operate, and other factors. The precise procedure selected for a patient was determined only after careful evaluation of all aspects. In the earlier years, the Billroth II gastrectomy was favoured, and gastro-

TABLE IV.—Complications Relative to Ulcer Type

Type of ulcer	*Perforation*		*Hemorrhage*		*Stenosis*	
	Number of cases	*Incidence*	*Number of cases*	*Incidence*	*Number of cases*	*Incidence*
		%		%		%
Penetrating duodenal ulcer	28	15.8	69	40.0	46	26.3
Non-penetrating duodenal ulcer	8	15.2	13	23.2	21	37.5
Duodenal and gastric ulcers	1	4.0	12	48.0	3	12.0
Gastric ulcer	—	—	24	40.7	5	9.1
Stomal ulcer	7	50.0	5	35.9	—	—

TABLE V.—Operations for Duodenal and Gastric Ulcers

Operation	Penetrating duodenal ulcer		Non-penetrating duodenal ulcer		Co-existing duodenal and gastric ulcers		Gastric ulcer	
Billroth I	2 (vagotomy, 1)	1.2%	12	21.4%	7	28%	30	50.8%
Billroth II	133 (vagotomy, 4)	77.3%	37	66.1%	18	72%	29	49.2%
Gastrojejunostomy or Finney	31 (vagotomy, 12)	18.0%	5 (vagotomy, 1)	8.9%	—	—	—	—
Vagotomy alone	6	3.5%	2	3.6%	—	—	—	—
Total	172		56		25		59	

jejunostomy was the procedure adopted for the aged and infirm, especially for relief of obstruction. Later, Billroth I resections were practised extensively, particularly for gastric lesions, and vagotomy proved a valuable addition to many other operations in the effort to eliminate anastomotic ulceration and its sequelae. With time and experience, it has been possible to make more intelligent, specific selections, bearing in mind the nutritional and psychological status of the patient, the presence of degenerative diseases, and the pros and cons of the different surgical methods; always aiming for lasting control of the acid-pepsin secretion and good gastric emptying, and seeking to avoid all forms of morbidity as well as mortality.

Operations

The operations actually performed for the various types of ulcer case are tabulated in Table V.

Resections were fairly standardized and radical, in general amounting to some 75%. The Billroth II type usually consisted of a short-loop, posterior, isoperistaltic anastomosis, according to the Hofmeister-Finsterer plan, with two layers of sutures (through-and-through chromic catgut and a serosal layer of fine cotton sutures, Cushing fashion). The stoma was made small routinely and attached below the transverse mesocolon. When the Billroth I method was employed, the resection was equally radical as a rule, and the stoma small, involving only part of the cut end of the stomach adjoining the greater curvature. Good mobilization of the duodenum was required. Gastrojejunostomy was generally posterior, with the same technical considerations as those enumerated above.

In each of the four Finney pyloroplasties, the distal limb of the incision was carried down in the duodenal wall practically to the level of the ampulla of Vater. Vagotomies were performed by both the thoracic and abdominal routes, care being taken to seek all fibres of the nerves; when no other reason for opening the abdomen existed, the upper approach was favoured.

The duodenal stump posed a special problem in certain of the resections dealing with a deeply penetrating, posterior duodenal ulcer. Fatal leakage in a single early case of this kind underscored the need for meticulous attention to this item. The exact management was individualized to suit the circumstances. In simple situations, a through-and-through layer of chromic catgut was followed by a layer of cotton sutures inserted according to the Cushing method, with a third layer of interrupted cotton sutures turning the closed stump over against the fascia overlying the pancreas. Frequently the resection extended into the margin of the ulcer, and local fixation and infiltration demanded ingenuity. Sometimes the Yudin type of closure solved the problem. In the worst cases, and as a last resort, a catheter was sutured simply into the end of the contracted and distorted stump by means of two or more purse-string rows, and brought out through the omentum for temporary drainage. The catheter was removed after 10 days or so without incident. Frequently one could anticipate trouble about a hazardous stump and avoid it by transection of the distal pyloric antrum, making certain to core out every bit of antral mucosa. Closure then could be effected with ease in the relatively healthy superficial gastric tissues, leaving the ulcer as

TABLE VI.—Background of Original Stomal Ulcers

Gastrojejunostomy	9	64.3%
Billroth I	1	7.2%
Billroth II	4	28.5%
Total	14	

TABLE VIII.—Antecedent Operation in all (30) Stomal Ulcers

Gastrojejunostomy	18	60.0%
Finney pyloroplasty	2	6.7%
Billroth I gastrectomy	3	10.0%
Billroth II gastrectomy	7	23.3%

a whole totally undisturbed. Some of the ulcer beds that were opened extended deeply into the retroperitoneal tissues, the pancreas, or even into the liver.

Associated conditions received surgical attention and correction in a number of patients. Appendectomy was practically routine. Some hernias of the incisional or umbilical type, lying in the field of operation, were repaired. Hiatus hernia repair was combined with gastrectomy in one patient and with vagotomy and gastrojejunostomy in another. Cholecystectomy was performed in 11 patients, a hydronephrotic right kidney was removed in one, and other occasional procedures were related to Meckel's diverticulum, ovarian cysts, etc.

ulcers is obvious. This fact will be attested to further by additional data related to the incidence of postoperative complications and sequelae presented later. Ulcer complications were unduly prevalent in this group – one or more being recorded in 22 patients, or 73.3% of those with stomal ulcers. The various operations performed for stomal ulcers in both groups are summarized in Table IX.

TABLE IX.—Operations for Stomal Ulcers

Type	*No. of patients*	%
Billroth II	17	56.6
Vagotomy	10	33.3
Miscellaneous	2	6.6
None	5	16.6

TABLE VII.—Acquired Anastomotic Ulcers in Series

Operation	*Stomal ulcers*	*No. of operations*	*Incidence*
Gastrojejunostomy	9 (vagotomy, 2)	36	25%
Finney pyloroplasty	2	4	50%
Billroth I gastrectomy	2	51	3.8%
Billroth II gastrectomy	3	234	1.28%
Vagotomy alone	—	16	—
Total	16	341 operations in 326 ulcer patients	

Anastomotic ulcers

The problem of anastomotic ulcers deserves special brief discussion. Fourteen patients presented with this lesion already established, following operations as listed in Table VI.

In 16 cases in the total series stomal ulceration was a later complication (proved in 11 and strongly suggested by the clinical and roentgenological evidence in the other five, see Table VII).

Adding these two groups together, we obtain the figures shown in Table VIII. The importance of simple drainage procedures in the production of anastomotic

As a rule subtotal gastrectomy was performed upon those patients who had only had a drainage procedure previously, and vagotomy was added where resection had already been carried out. Revision resection was performed in several patients in whom the original gastrectomy appeared to have been inadequate or faulty. Perforation of a stomal ulcer was diagnosed twice and treated surgically, once after a Billroth I type gastrectomy and once after gastrojejunostomy with vagotomy. In view of the fact that five of the 30 patients with anastomotic ulceration received only medical management, it will be evident that more than one operation was carried out in four cases.

TABLE X.—Postoperative Complications and Sequelae

Wound problems:		
sepsis, etc.	18	
dehiscence	4	6.7%
Abdomen:		
subphrenic abscess	2	
acute pancreatitis	1	
intestinal obstruction	2	2.4%
strangulated hernia	1	
perforation of stomal ulcer	2	
Chest:		
atelectasis, etc.	3	0.9%
Phlebitis and embolism	5	1.5%

Postoperative Complications and Sequelae

The significant postoperative problems in relation to the entire series are set out in Table X under appropriate headings. These do not require detailed comment. Wound complications arose more frequently than one likes to see and in several instances removal of unabsorbable suture material from the wounds was necessary before satisfactory healing was obtained. Dehiscence occurred four times; this was equally divided between the right upper paramedian and the midline incisions which had been closed by use of multiple layers of unabsorbable material (chiefly fine stainless steel wire or cotton interrupted sutures). In each case secondary closure was effected by means of interrupted, through-and-through, heavy braided silk sutures inserted in figure-8 fashion. Chest complications were not troublesome, but special care was devoted to the elimination of bronchial secretions and to oral hygiene. Similarly, every effort was made to prevent thromboembolic phenomena by maintenance of active general and local blood flow and all other helpful measures. In spite of this care, the problem was recognized five times, minor embolization occurring twice and fatality once. In one case acute pancreatitis developed rather suddenly on the fifth day; the clinical features suggested acute peritonitis in the upper abdomen and laparotomy was performed promptly in the mistaken belief that leakage had occurred at the suture line. Typical areas of fat necrosis were apparent. Acute small bowel obstruction developed 16 days after gastrectomy in one patient and some weeks after gastrojejunostomy in another case; in each of these a mechanical block was caused by adhesions. In one elderly patient, an associated inguinal hernia became strangulated before discharge from hospital. The two instances of perforation of stomal ulcers have already been mentioned, both occurring many months subsequent to the original procedures. One of them developed a subphrenic abscess which necessitated drainage; this same complication followed gastrectomy in a patient with a penetrating duodenal ulcer.

All available evidence pointed to postoperative anastomotic ulceration complicating the various operations of this series in 16 cases, as shown in Table VII. The high incidence following pyloroplasty and gastrojejunostomy when not protected by vagotomy is shown. Two cases were not protected by this procedure, but these were in the early days of vagotomy; were probably incomplete, and it is felt that they were not selected to best advantage. The extremely low incidence following resection is striking, as is the complete protection afforded by vagotomy combined with resection.

Postoperative Mortality

Eight hospital deaths occurred in this series. The data relevant to these are presented in compact form in Table XI, indicating the type of ulcer, the age of the patient, the operation carried out, the day of demise and the cause as far as could be ascertained. Cardiovascular and renal diseases predominated. Very early in the series there was one fatality from peritonitis due to leakage of the duodenal stump. One man jumped suddenly from an upper window on the fifth postoperative day landing on concrete below, disrupting both his cranium with its contents and his abdominal wall. His suicide was totally unaccountable.

A tabulation of the fatalities according to the operation performed is set forth in Table XII. The notably high mortality which attended the simpler procedure of

TABLE XI.—Causes of Death

Patient	*Age*	*Type of ulcer*	*Operation*	*Postoperative day*	*Cause*
C.P.	54	Penetrating duodenal ulcer	Billroth II	2	Peritonitis
G.T.	37	Penetrating duodenal ulcer	Billroth II	5	Suicide
L.A.	51	Penetrating duodenal ulcer	Gastrojejunostomy	14	Intestinal obstruction and uremia
E.H.	76	Penetrating duodenal ulcer	Gastrojejunostomy	8	Myocardial infarction?
H.C.	73	Duodenal and gastric ulcers	Emergency Billroth II	14	Cerebrovascular accident?
J.C.	55	Duodenal ulcer, stenosing	Billroth II	2	Embolus
J.W.	87	Gastric ulcer	Billroth I	10	Pneumonia, pyelonephritis
W.L.	76	Duodenal ulcer, stenosing	Gastrojejunostomy	21	Uremia

gastrojejunostomy is readily explained by the fact that this was utilized commonly in the poor-risk elderly patients, who had a variety of associated degenerative conditions. The concurrent surgical removal of certain other diseased viscera did not affect the statistics adversely as far as could be determined.

TABLE XII.—Fatalities According to Operation

	Patients	*No. of deaths*	*Incidence*
			%
Billroth I	in 51	1	1.96
Billroth II	in 234	4	1.70
Gastrojejunostomy	in 36	3	8.3
Vagotomy	in 18	0	0
Case mortality	8 patients of 326		2.4

Late Results

The postoperative results have been judged critically, by the patient's own doctor in the vast majority of cases, and the processed material is summarized in Table XIII. An attempt has been made to evaluate the end results with regard to the variety of ulcers and the operative management. Those categorized as "good" have been free from significant digestive symptoms and disability; showed quite satisfactory nutritional status, and were able to work steadily. The group classified as "fair" complained of some gastric or bowel disturbances, significant dumping distress or other minor disabilities, or showed defective nutrition. A "poor" result meant recurrent ulceration, marked complaints, real disability, or other factors that prevented normal life and work. Several of the patients who fell into this unfortunate class were chronic alcoholics while others showed major psychiatric problems.

In retrospect, it would seem that these were the outstandingly bad results of medical treatment at an earlier stage; that they never co-operated fully, and that no surgical measures could compensate for their innate inadequacies. It is regrettable that a number of patients have been lost to follow-up study. Several of these gave early promise of success, and one knows that in general the results following gastrectomy appear to improve with the passage of time (to some extent in contradistinction to those following gastrojejunostomy, since stomal ulceration often appears many years later); these few could not be classified, but they suggest that the real outcome may have been even better than the figures quoted.

Comment

A survey of the cases of stomal ulcer reveals that an astounding variety and number of operations were performed upon these patients. Some of these operations were ill-advised procedures, obviously directed at organs that were not responsible for the symptoms (appendix, biliary tract, and hernias of the epigastric and umbilical types in particular). Others were inadequate in concept, e.g. gastrojejunostomy for active duodenal ulceration in patients of any age. The results in still others illustrated the dreadful effects of faulty surgical judgment or technical ineptitude, e.g. the jejunum was anastomosed high up to the body of the stomach in one patient, and in two young men anastomosis had been effected between the stomach and the ileum in one case and to the transverse colon in the other. The disastrous effects of such so-called surgery need not be detailed. One of these unfortunate men had

TABLE XIII.—Postoperative Results

Condition	Operation	Good	Fair	Poor	Inadequate follow-up	Total
Penetrating duodenal ulcer	Billroth I	1 (50%)	—	1 (50%)	—	2
	Billroth II	104 (78.2%)	17 (12.8%)	4 (3.0%)	8 (6.0%)	133
	Gastrojejunostomy and Finney	14 (45.1%)	3 (9.8%)	13 (41.9%)	1 (3.2%)	31
	Vagotomy	5 (83.3%)	1 (16.7%)	—	—	6
						172
Non-penetrating duodenal ulcer	Billroth I	8 (66.7%)	2 (16.7%)	1 (8.3%)	1 (8.3%)	12
	Billroth II	26 (70.3%)	4 (10.8%)	3 (8.1%)	4 (10.8%)	37
	Gastrojejunostomy and Finney	1 (20.0%)	1 (20.0%)	3 (60.0%)	—	5
	Vagotomy	1 (50.0%)	1 (50.0%)	—	—	2
						56
Duodenal and gastric ulcers	Billroth I	5 (71.4%)	1 (14.3%)	—	1 (14.3%)	7
	Billroth II	10 (55.6%)	2 (11.1%)	4 (22.2%)	2 (11.1%)	18
						25
Gastric ulcer	Billroth I	20 (66.7%)	3 (10.0%)	4 (13.3%)	3 (10.0%)	30
	Billroth II	21 (72.4%)	4 (13.9%)	1 (3.4%)	3 (10.3%)	29
						59
Stomal ulcer	Billroth II	12 (70.6%)	4 (23.5%)	1 (5.9%)	—	17
	Vagotomy	5 (50.0%)	4 (40.0%)	1 (10.0%)	—	10
						27

three further laparotomies for his misery and bowel obstructions before my resection, which restored him to full comfort and has enabled him to run his own business for the last 15 years. A selection of nine patients who figure ultimately in the favourable group, mainly of those treated for anastomotic ulcers, reveals an astonishing total of 43 different operative procedures. The definitive operations are included in the final figure.

Three patients with deeply penetrating, long-standing duodenal ulcers had received so much inadequate care or actual mismanagement, both medical and surgical, that they exhausted several doctors, had become more or less dependent on narcotics, and were regarded as functional problems. Very often the true situation can be suspected promptly if one is willing to go back with an open mind and review carefully and critically the early phases of the history. One man had a typical story of major and minor periodicity of his distress for 20 years, followed by eight years of constant misery and incapacity, during which time four abdominal operations had not helped him. After a correct assessment and resection he made a most gratifying recovery, and even his drug dependence cleared up readily. The other cases illustrated the same principles.

It has been claimed that late deaths after major gastric surgery are more common than most people appreciate. The follow-up study in this series has brought to light 20 such late deaths, the time interval from operation varying from under one year to nearly 19 years. In seven cases the cause was cardiovascular and in five malignant disease was responsible. Miscellaneous causes accounted for the remaining eight. Many of these patients were quite elderly. It is not possible to draw any significant conclusions from this small group.

Summary and Conclusions

A personal series of 326 gastroduodenal ulcer patients has been analyzed and the essential findings have been tabulated and discussed.

Various problems have received special attention, including the chief ulcer complications, diagnostic difficulties and technical management of the deeply eroding lesion.

An attempt has been made to compare the different operative methods utilized for various situations in relation to morbidity, mortality and effectiveness.

Anastomotic ulceration has been considered in some detail.

Certain unfavourable groups can be recognized: those with multiple ulcer complications; intense symptoms and intractability; the younger age groups, and chronic alcoholics or emotionally unstable personalities. Resort to definitive surgery in these suggests the wisdom of combining vagotomy with another procedure such as Billroth II gastrectomy.

In general, individualization of management at all stages is most important and assessment must include the physical and psychological status of the individual as well as evaluation of the ulcer and its complications. Feeble and malnourished persons are not good candidates for radical resection. The choice here would seem to be gastrojejunostomy and vagotomy.

Radical resection is indicated only rarely in the presence of perforation of a duodenal ulcer, but should be considered in the event of multiple perforations. Two such resections were performed in this series with a successful outcome in each.

Résumé

Dans cet article, 326 cas d'ulcères gastro-duodénaux ont été analysés et étudiés; les résultats principaux sont rapportés sous forme de tableaux (distribution selon le sexe, selon l'âge, index d'incidence des complications, types d'opérations employées, etc.).

Jadis l'intervention la plus pratiquée était la gastrectomie selon Billroth 2ème manière, tandis que la gastro-jéjunostomie était réservée aux malades âgés pour pallier aux symptômes de sténose. Plus près de nous, les résections type Billroth I sont maintenant faites de façon intensive. La vagotomie se révèle être un précieux adjuvant pour contrecarrer l'apparition de l'ulcère peptique; elle se fait indifféremment par voie thoracique ou abdominale, selon les cas. Dans la présente série, l'indice de mortalité se monte à 2.4%, pour ce qui est des morts dues à l'opération elle-même. Les résultats éloignés ont pu être étudiés de façon approfondie grâce à la collaboration des médecins traitants; ces résultats sont présentés ici en détail, catalogués par maladies et par type opératoire. Les groupes défavorables sont: les ulcères multiples; les ulcères accompagnés d'une symptomatologie bruyante; les alcooliques chroniques et les patients spécialement émotifs ou instables. Les résultats les plus favorables semblent être ceux du Billroth II associé à la vagotomie. Il faut cependant se garder de vouloir appliquer à tous un traitement uniforme; le choix de la tactique opératoire dépendra de l'ensemble des conditions physiques et psychologiques du malade. La résection radicale totale n'est qu'exceptionnellement indiquée.

THE SURGICAL TREATMENT OF INTRACRANIAL MENINGIOMAS. Collin S. MacCarty, Consultant in Neurologic Surgery, The Mayo Clinic, and Associate Professor of Neurologic Surgery, The Mayo Foundation Graduate School, University of Minnesota, Rochester, Minn. 69 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $5.00.

This small monograph was prepared from a review of Dr. MacCarty's experience with meningioma at the Mayo Clinic and presented to the Congress of Neurologic Surgeons in Miami in October 1959.

The author has confined himself to the surgical treatment of these tumours, thus limiting the scope of interest of the manual to neurosurgeons, particularly those in training. Operative notes and drawings from actual case histories have been used to emphasize points of technique. Each chapter deals with the operative technique for one of nine different sites of these formidable tumours. Emphasis has been placed on the proper planning of incision and bone flap so as to have reasonable control of the situation from the beginning.

A graphic description is given of the recovery of a woman who was quadriplegic and mute after resection of a tumour-occluded sinus at or behind the Rolandic region. The author rightly cautions against the ligation of the sinus posteriorly, for in most instances the patient would not be so fortunate.

The drawings of the convexity and parasagittal tumours are excellent. Three-dimensional or shadow drawings of the basal tumours might have emphasized further the caution with which many of these lesions must be approached in order to avoid injury to cranial nerves and major arterial channels.

CARCINOMA AMONG LABRADOR ESKIMOS AND INDIANS

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IT HAS been said that there is no carcinoma among the Eskimo population. Hildes[1] in a recent article quotes various authors who present evidence which does not support this theory.[2, 3]

This review reports our personal experiences over the 10-year period of 1950-1960 with cases of carcinoma among the full-blooded Eskimo and Indian population of Northern Labrador.

All of these cases, except one, were seen by the author at the Grenfell Hospital, St. Anthony, Newfoundland, the majority being referred by Dr. Anthony Paddon of the Grenfell Hospital at North West River, Labrador. The one exception, who was seen by the author, was a 50-year-old Indian woman living in a tent in the middle of Labrador. This patient had a rodent ulcer of the face, which she had had for many years and about which she had never before seen a doctor.

As there is no other medical agency working in this area and because the natives, particularly the Eskimos, are under close medical supervision by the International Grenfell Association, it is logical to assume that there is little likelihood of a case of carcinoma in an Eskimo in the area escaping detection. The problem is different in the case of the Indians, many of whom remain nomadic and difficult to keep under constant observation, and who are still somewhat reticent about seeking medical care. Therefore, it is possible that some cases of carcinoma among the Indians might not be seen.

There are 850 Eskimos and 370 Indians living in Labrador North, i.e. that area of Labrador north of Lake Melville and Hamilton River.[4]

The Eskimos studied lived in four main communities: Hopedale, Nain, Nutak and Hebron. The Indians, a group of the Montagnis, make their headquarters during the summer months at North West River and at Davis Inlet.

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Following the first two years of cancer reporting and based on the Newfoundland census figures for 1956, the general incidence of carcinoma in the province of Newfoundland and Labrador reported in 1958 was 1.9 per 1000 population.[5]

During the 10-year period, 13 Eskimos were seen with proved malignancies and two others with presumed malignancies which were not proved pathologically. These two are not considered statistically in this paper. During the same period three Indians with carcinoma were observed. A yearly incidence of 1.5 per 1000 of population among the Eskimos and a yearly incidence of 0.9 per 1000 of population among the Indians were seen, the figure for the Eskimos being only slightly lower than that for the whole population of the province of Newfoundland. In view of the small numbers of the Indians and the likelihood that some of the cases among them might have been missed, the incidence among these natives is of no true value except to show that carcinoma does occur in the Labrador Indians.

The sites of carcinoma were as follows:

TABLE I.—ESKIMOS

Bone	1
Pharynx	1
Stomach	1
Breast	1
Pancreas	1
Large bowel	4
Bladder	1
Cervix	3
Total	13

TABLE II.—INDIANS

Parotid gland	1
Rectum	1
Rodent ulcer on face	1
Total	3

Three of the Eskimos are alive and well one to ten years after treatment and one Indian remains alive; this is the patient with the rodent ulcer.

TABLE III.—Eskimos

Patient	Chart No.	Age (years)	Sex	Home	Year	Diagnosis	Treatment	Outcome	Remarks
D.J.	22810	15	M	Hebron	1950	Osteogenic sarcoma, right leg	High amputation right leg.	Alive	Curative surgery attempted.
L.O.	23246	50	M	Nain	1951	Carcinoma, bladder	Suprapubic cystotomy and fulguration of tumour.	Expired (March 3, 1951)	Palliative surgery. Postmortem showed metastasis from bladder to liver and abdominal glands. Pulmonary T.B.
E.W.	23627	26	F	Rigolet	1951	Carcinoma, cervix	E.U.A., D & C, biopsy cervix. Radium inserted.	Expired (Nov. 23, 1951)	August: "frozen pelvis", radioresistant.
M.L.	24052	61	M	Nain	1951	Carcinoma, ampulla, duodenum	Exploratory laparotomy and cholegastrectomy.	Expired (Nov. 22, 1951)	Palliative surgery only. Postmortem not done.
E.T.	24136	25	F	Nutak	1951	Carcinoma, cervix	D&C and biopsy. Radium inserted. Total 6000 mg. hrs.	Expired (1953)	Palliative surgery only.
S.N.	25763	61	M	Hebron	1953	Carcinoma, cecum with metastasis	Exploratory laparotomy and Witzel enterostomy.	Expired (Oct. 12, 1953)	Palliative surgery only.
L.O	27563 30362	71	F	Nutak	1955 1957	Carcinoma, colon	Right hemicolectomy	Expired (Nov. 5, 1957)	Readmitted 1957—Diagnosis carcinoma colon, post-resection —hepatic metastasis. Palliative surgery only.
C.K.	29455	53	F	Nain	1957	Infiltrating duct carcinoma, breast	Right radical mastectomy. Deep x-ray therapy postoperatively.	Alive and well	Readmitted 1960. Curative surgery attempted.
G.B.	27063	66	M	Nain	1955	Carcinoma, cecum	Exploratory laparotomy and ileostomy.	Expired (Feb. 7, 1955)	Palliative surgery only. Postmortem showed: (1) carcinoma ileocecal valve; (2) generalized peritonitis.
J.F.	29242	60	M	Nain	1956	Epidermoid carcinoma, right tonsil	Radium needles	Expired (Nov. 9, 1956)	Palliative surgery only. Postmortem showed death due to cerebral embolism.
J.F.	32090	38	F	Hopedale	1959	Carcinoma, cervix	Hysterectomy	Alive	Curative surgery attempted.
B.J.	32604	59	M	Hopedale	1959	Carcinoma, stomach	Anterior gastro-enterostomy for inoperable carcinoma.	Expired (November 1959)	Palliative surgery only.
T.T.	32989	69	M	Nain	1959	Adenocarcinoma, middle descending colon	Exploratory laparotomy and colostomy.	Expired (April 1960)	Palliative surgery only.

TABLE IV.—Indians

Patient	Chart No.	Age (years)	Sex	Home	Year	Diagnosis	Treatment	Outcome	Remarks
J.P.	28259	46	M	Davis Inlet	1956	Carcinoma, parotid gland	Radical neck dissection, right; excision, carcinoma parotid gland.	Expired (February 1956)	Palliative surgery only. Expired presumably because of cerebral embolus.
J.R.	27019	72	M	North West River	1958	Adenocarcinoma, rectum	Abdomino-perineal resection.	Expired	Palliative surgery, tumour extended into bladder.
M.A.	—	80	F	North West River	1959	Rodent ulcer of face	No treatment	Alive	No surgery

A summary of these cases is shown in Tables III and IV.

In view of the interest expressed by many as to whether or not Eskimos ever develop carcinoma of the breast, the following case history is noteworthy.

Case Report

C.K., a 53-year-old Eskimo woman from Nain, Labrador, was admitted to the Grenfell Hospital, St. Anthony, Newfoundland, in January 1957 complaining of "funny feelings" in her right arm associated with weakness. In

December 1956 she noticed a lump in her right breast and in her right axilla, which she described as being tender. Physical examination revealed an Eskimo woman who spoke no English, who was well nourished and in good physical condition. A nodule, 2½ x 1½ x 1½ cm., was felt in the lower outer quadrant of the right breast and was freely movable. There was a palpable node in the right axilla.

A radical mastectomy was carried out on January 14, 1957, and the following pathological report on the surgical specimens was provided by Dr. J. E. Josephson, pathologist, St. John's, Newfoundland:—

Gross description.—The gross specimen consists of the right breast removed by radical mastectomy. It measures 12 x 10 x 5 cm. and is composed superficially of an elliptical-shaped piece of skin measuring 14 x 6.5 cm., in the centre of which is a fairly prominent nipple. It has been previously incised revealing a large mass of firm, glistening white tissue spreading throughout the fatty and parenchymal-like tissue of the breast. Just inferior to the nipple there is an irregularly outlined mass of firm, hard, dense, white tissue which appears to be radiating to the surrounding breast tissue. A number of shaggy irregularly outlined pieces of tissue from the axilla accompany the breast. These have been previously incised revealing that they contain a number of lymph nodes, several of which appear to have been infiltrated by the surrounding fatty-like tissue.

Microscopic report and remarks.—This is a scirrhous type carcinoma in which much of the breast tissue is composed of dense avascular collagenous connective tissue which is being infiltrated by small knots and occasional glandular structures composed of neoplastic epithelial cells. These cells have rounded nuclei which show considerable variation in size and shape. The chromatin content also varies considerably from cell to cell. The cytoplasm is pale and generally well outlined. Mitotic figures within the tumour mass are scanty. Sections taken through the accompanying axillary lymph nodes reveal that these show prominent reactive hyperplasia with some dilatation of the cortical and medullary sinuses, accompanied by some fibrosis. No evidence of tumour is present in these nodes, or in the fatty tissues around them.

Diagnosis.—Infiltrating duct carcinoma of breast; chronic axillary lymphadenitis.

The patient made an uneventful recovery and in April 1957 was treated in St. John's, Newfoundland, with postoperative deep radiation. She remains alive and well to date with no evidence of metastasis.

It is interesting that there were no cases of carcinoma of the skin or lip among the Eskimos. Carcinoma of the lip is the commonest malignant lesion seen in the white population here and this is felt to be due to exposure, pipe-smoking and the holding of tarred twine in the mouth while making and mending fish-nets. The problem of early diagnosis is quite apparent. Because of the extent of the lesion when seen in 10 of the 13 Eskimos reported, palliative surgery or no surgery at all was attempted. No curative surgery was attempted in any of the Indian patients. Three of the Eskimos had definite curative surgery attempted and, interestingly, all three remain alive and well. An educational program is as important among the native population as it is among the general population, but it is difficult to carry out because of language and social problems.

Summary

Thirteen proved cases of carcinoma were seen in a small group of Labrador Eskimos over the 10-year period, 1950-1960.

There were three survivors in the Eskimo group.

Three cases of carcinoma were seen in a small group of Labrador Indians during the same period.

Acknowledgment

The author wishes to express his thanks to Miss Jane Macdonald, R.N., for following up the patients reported in this paper.

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Résumé

On a prétendu que le cancer ne se rencontre jamais chez les populations esquimaudes. Le présent article vient infirmer cette assertion, passant en revue les cas de carcinome observés chez les Indiens et les Esquimaux du Nord du Labrador pendant une période de 10 ans (entre 1950 et 1960).

La population qui fut étudiée comporte 850 Esquimaux et 370 Indiens. Les cas de cancer qui furent découverts se répartissent comme suit: os, pharynx, estomac, sein, pancréas, gros intestin, vessie, et col utérin chez les Esquimaux; glande parotide, rectum et face chez les Indiens.

L'incidence annuelle de carcinomatose se monte à 1,5 pour 1000 chez les Esquimaux et à 0,9 pour 1000 chez les Indiens. Il est à noter que le cancer de la peau ou de la lèvre n'a jamais été observé.

Un cas de cancer mammaire est décrit en détail.

BASAL CELL CARCINOMA*

STUART GORDON, M.S., F.R.C.S.[C], F.A.C.S., *Toronto*

Basal cell carcinoma (rodent ulcer) is a form of malignancy which may develop in any place on the surface of the body except the palms of the hands and the soles of the feet,[5] but, in fact, it appears in about 90% of instances on exposed surfaces of the body, mainly the face (Fig. 1). It is proposed to review the features of 115 patients, 75 men and 40 women, representing 123 lesions treated by the author. Lesions which might have been induced by radiation were excluded.

Untreated, the basal cell carcinoma gradually, insidiously and relentlessly destroys tissues as it advances, until it reaches some structure whose invasion is incompatible with life. Metastases from basal cell carcinoma are very rare but have been reported.[3,8] In spite of the fact that the lesion develops on the body's surface, usually on an exposed portion, and spreads by direct extension, some 6% of deaths from carcinoma are due to carcinoma of the skin.[7] It is obvious that any lessening of the mortality and morbidity figures must be the result of earlier diagnosis and more adequate treatment.

It would seem that there is little doubt that the blue-eyed blonde is more apt to develop this disease than the brunette who tans easily.[2,5] Schrire[9] states that the ratio of Europeans to Negroes who develop rodent ulceration in South Africa is 27 to 1. Although commonest in the fifth and sixth decades, it may occur at any age and has

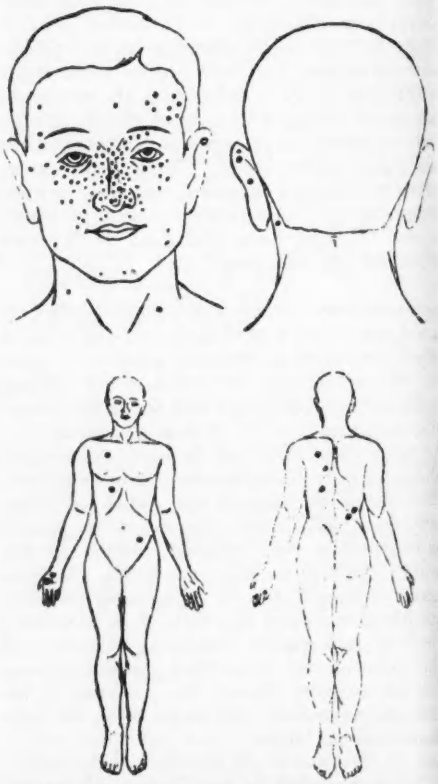


Fig. 1.—Chart indicating site of lesions discussed in the text.

*Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Ottawa, January 1961.

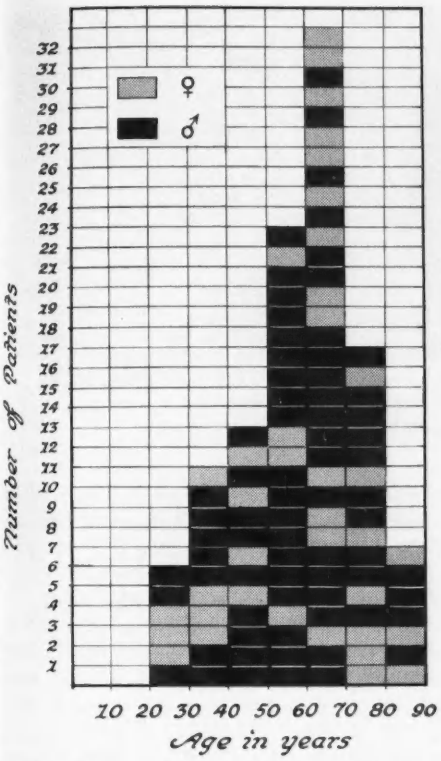


Fig. 2—The age distribution is shown, the lighter squares representing women, the darker, men.

been reported in the very young. Macomber, Wang and Sullivan[6] recorded its occurrence in a girl of 14 years. The average age in both men and women of this group was just over 57 years (Fig. 2).

The disease occurs in three forms. The commonest develops as a yellowish-grey, waxy nodule in the skin (Fig. 3). Eventually a crust forms in the central portion. This crust falls off or is knocked off and another forms. Growth is slow. The lesion may have been present for from three months to many years before treatment is sought. Stretching the skin about the lesion reveals a semi-translucent, ring-like thickening with a depressed central area, often termed the "greasy doughnut".

The second form, the cystic basal cell carcinoma (Fig. 4) is less common and appears less malignant than the other two. Originating in the deeper layers of the epidermis, it tends to extend more in the

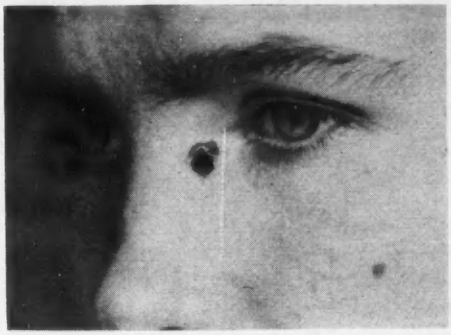


Fig. 3.—Typical appearance and site of a basal cell carcinoma. The patient is a blue-eyed blonde woman, aged 24 years.

direction of the underlying tissues than to the surface and for this reason has been called the "iceberg tumour". It is always primarily a lesion of the skin. It apparently extends more slowly than the other two forms.

Form number three is rare, the pigmented rodent ulcer (Fig. 5). This type of tumour was noted nine times in 200 cases examined at the New York Hospital.[5] It occurred twice in the series described in this report.

Rodent ulcer may be confused with senile keratosis, molluscum sebaceum (Fig. 6), and squamous cell carcinoma (Fig. 7). There is really only one way of establishing the identity of these lesions and that is by biopsy. Any condition of the skin, the nature of which is doubtful, should be submitted to microscopic examination; this dictum applies not only to a lesion of



Fig. 4.—A cystic basal cell carcinoma. It is typical in both conformation and site.

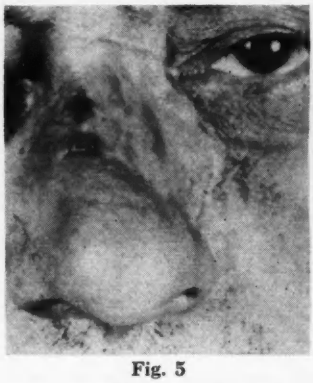


Fig. 5

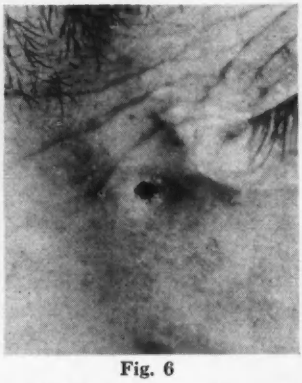


Fig. 6



Fig. 7

Fig. 5.—A recurrent pigmented basal cell carcinoma of the nose. **Fig. 6.**—This molluscum sebaceum was excised in the belief that it was a rodent ulcer. **Fig. 7.**—This lesion was diagnosed as a kerato-acanthoma. It proved to be a squamous cell carcinoma.

which one is suspicious but to any whose character changes in any way, objectively or subjectively.

Treatment

There are three methods of treatment in more or less common use today, excision, radiotherapy and electrodesiccation. A fourth form of treatment, dermabrasion, has been suggested recently[4] and mention is made of it here only to state that, in my opinion, dermabrasion has no part to play in the treatment of epithelial malignancy. It is this kind of inadequate treatment that can only result in too high a proportion of failures when applied to any form of malignancy.

As the lesion is superficial it lends itself to early diagnosis and effective local treatment; as it is local, complete ablation is possible when diagnosis is made early.

As stated previously no lesion of the skin should be treated without a definite diagnosis and this can be obtained only by biopsy, a surgical procedure entailing the removal of a pie-shaped or canoe-shaped portion which includes a full-thickness section of the lesion, with subcutaneous tissue and adjacent skin. Tiny scooped-out bits of a lesion do not constitute a true biopsy and their use may lead to incorrect interpretation of the nature of the disease and so render valueless any study which includes reports obtained in this manner.[1] Why then not adopt as a routine the excision of the lesion, along with an area of skin and subcutaneous tissue sufficient to ensure complete removal of the lesion, check the adequacy of the excision by quick section and close the defect by whatever method is appropriate? Fifty-six of these patients required more than direct closure for repair. Twelve were repaired by a split-skin graft; seven with a free full-thickness skin graft, usually postauricular; 31 were closed by some form of local tissue shift; and in six patients a combination of these procedures was used. If a quick section is not available, and the pathologist's routine report indicates inadequate removal, then re-excision, with apologies to nobody, must be carried out. Eighty-eight patients were treated primarily by this method, although quick section was not done in the earlier cases. Of 34 patients whose primary treatment consisted of excision checked by quick section, there was only one recurrence. No further record can be obtained in 18. Eight died of some other cause, without recurrence of rodent ulceration, in from one to six years, and two died of their disease. Four are alive with proved recurrence. Fifty-six have remained alive and well for periods ranging from months to 28 years.

Rodent ulcers that occur on the cheeks, as in any area that does not overlie cartilage, will respond well to expert radiation therapy in many cases. The same is not true of lesions over cartilage, [6, 7] for example, those on the ears, eyelids, and the

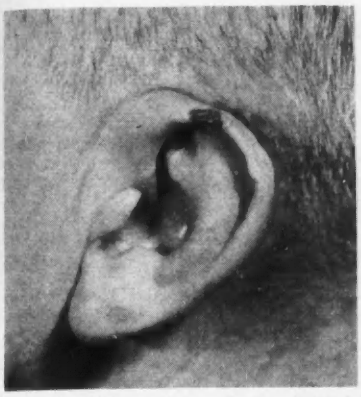


Fig. 8.—Radiodermatitis and chondritis following radiation therapy of a rodent ulcer nine months previously.

lower part of the nose. Many of these if treated by radiation develop a chondritis (Fig. 8) and require surgical intervention later; so why not, when presented with a basal cell carcinoma in these areas, resort to surgical excision at once?[3] Twenty-one of the patients in this series presented for treatment of a recurrence following radiation therapy. Twelve of these have remained well for periods of from one to 11 years following single excision; four had a second recurrence which was again excised and they have since remained well; two died of their disease, and three have been lost to follow-up study.

If a rodent ulcer responds initially to adequate radiation and later shows evidence of recurrence or spread, it should never be radiated again but should automatically become a surgical responsibility.[3, 8, 10, 11] It has been demonstrated that this lesion is not really radiosensitive and cannot be controlled by well-supervised radiotherapy.

Electrodesiccation has less to offer than either excision or radiation (Fig. 9). The limits of tissue destruction are difficult, if not impossible, to define. A biopsy is necessary to establish the diagnosis and if this lesion is of a size suitable for this form of therapy it will lend itself readily to surgical excision.

A basal cell carcinoma should be treated the moment there is the slightest suspicion of its presence. All such lesions should be widely excised and checked, where possible, by both quick section and paraffin section, and the resultant defect closed by whatever means seems most appropriate; direct suture, split-skin graft, full-thickness skin graft, local or distant pedicle graft, or any combination of these procedures.

Patients who have been treated for rodent ulcer should be seen at regular intervals for the rest of their lives. A lesion, recurrent or new, as has been noted in many reports, may appear long after the arbitrary limit of five years has passed.

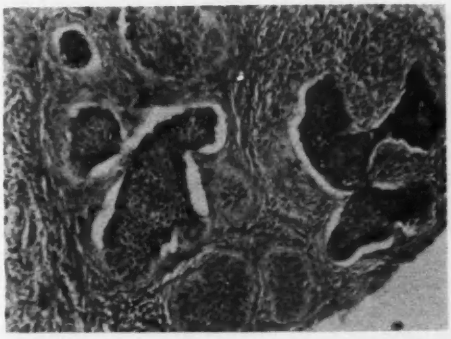


Fig. 9.—A proved rodent ulcer just medial to the medial canthus of the right eye was treated by electrodesiccation four months after onset. Two months later sections of the area after excision revealed the persisting presence of basal cell carcinoma. x 150.

Finally, in those unfortunate patients in whom recurrent disease has advanced to such an extent that any attempt at surgical treatment would result in marked deformity without any real assurance of cure, it is more humane to withhold one's hand. Symptomatic treatment with, perhaps, palliative radiation is now the only wise form of therapy. Palliative treatment in this instance is directed more at attaining relative mental peace than at affecting the actual disease.

Summary

Basal cell carcinoma is a malignant lesion occurring usually on the face. It rarely metastasizes but spreads by direct continuity. Early wide excision, the extent of which is checked by the pathologist, is

believed to be the best treatment. Regular re-examinations should be made for the remainder of the patient's life.

ACKNOWLEDGMENT

I wish to express my thanks to the Department of Medical Art, Department of Veterans Affairs, Sunnybrook Hospital, Toronto, for their help with the illustrations in this paper.

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RÉSUMÉ

Le carcinome cutané à cellules basales (ulcus rodens) se recontre dans n'importe quelle localisation sauf dans la paume des mains et la plante des pieds. Pour ce qui est des autres localisations, 90% des cas sont faciaux.

L'article présenté ici étudie 115 malades vus par l'auteur, dont 75 hommes et 40 femmes. Lorsqu'il n'est pas traité, le carcinome cutané basocellulaire grandit lentement, rongeant les tissus environnants, jusqu'à ce qu'il atteigne une taille incompatible avec la vie. Les métastases, bien que possibles, sont très rares. La maladie frappe de préférence les blonds, et l'on sait que le rapport d'incidence entre les Blancs et les Nègres est de 1 à 27 respectivement. L'âge moyen des patients, hommes ou femmes, est 57 ans et au-dessus.

Cette carcinomatose peut se présenter sous trois formes principales: soit comme un petit nodule cutané, de couleur gris-jaunâtre, parfois recouvert d'une croûte; soit comme un petit kyste, dont le développement est très lent et se fait vers les parties profondes de la peau; soit enfin, sous forme d'une petite tumeur pigmentée.

Le diagnostic différentiel est à faire avec le carcinome spinocellulaire, avec les kératoses et le molluscum. Le plus sûr moyen diagnostique est évidemment la biopsie.

Le traitement a été discuté: excision, radiothérapie, électrocoagulation, abrasion. L'auteur recommande une excision large, emmenant tous les tissus suspects, vérifiée par des coupes histologiques extemporanées. Ultérieurement, il y a lieu de surveiller le malade à intervalles réguliers pour prévenir les possibilités de récidive.

SURGICAL DISEASES OF THE LUNG. Buford H. Burch, M.A., M.D., Chief of Thoracic Surgery, Chest Center, Patton State Hospital, Patton, Calif., and Arthur C. Miller, M.S., M.D., F.A.C.S., Division of Surgery, College of Medical Evangelists, Loma Linda, Calif. 128 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $8.50.

This readable monograph is an excellent account of the personal experience of two surgeons. They set out to present to the reader a factual and brief account of the major surgical diseases affecting the lung. They have achieved their object. The format is informal and somewhat dogmatic in that it presents the attitudes of one group without clouding the field with conflicting opinions—but this cannot be taken as a criticism. Although brief, it is full of meat and contains an excellently reproduced group of radiographs at the end of the book to illustrate the text. The book can be read and digested easily in an evening and should appeal especially to medical students and practitioners, to whom it can be highly recommended. To medical students in particular, this volume will suffice to supply them with all the information they need on the surgical approach to diseases of the lung. The authors' wish was to give us a short and pleasant journey through this book and they have achieved this object as well as imparting a goodly amount of valuable clinical information and experience.

CASE REPORTS

MASSIVE HEMORRHAGE DUE TO DIVERTICULAR DISEASE OF THE COLON: A Case Illustrating the Bleeding Point*

I. SALGADO, M.D., G. K. WLODEK, M.D., W. H. MATHEWS, M.D. and H. ROCKE ROBERTSON, M.D., F.R.C.S.[C], F.R.C.S.(E.), F.A.C.S., *Montreal*

It is commonly stated that massive bleeding can arise from a diverticulum of the colon. In a number of reported instances[1-9, 11-15] in which bleeding has not ceased after institution of conservative treatment, resections of the involved colon have been carried out. While diverticula, with or without infection, have in each case been demonstrated in the surgical specimens, and while superficial ulceration has occasionally been observed,[3, 5, 6, 10-15] we have not discovered a single reported case in which the actual bleeding point has been demonstrated.

By chance, in a case that we encountered recently, our attention was directed to one of many diverticula in a patient who had bled massively, by the fact that a thrombus was seen to be protruding through the ostium of the diverticulum (Fig. 1). Serial sections through this particular diverticulum revealed the eroded vessel (Figs. 4 and 5).

Case Report

Mrs. S.S., a 79-year-old white woman, was admitted to The Montreal General Hospital at 5:00 a.m. on July 26, 1960. Her history revealed that for two weeks before admission she had had daily rectal bleeding and had been passing gross blood per anum during the preceding 24 hours.

In 1954, she was seen in the outpatient department when a diagnosis of arteriosclerotic heart disease and hypertension was made (blood pressure 210/110 mm. Hg). In 1956 she began to complain of increasing constipation and intermittent rectal bleeding. Diverticulosis of the sigmoid colon was discovered and repeated radiographs since that time have shown progression of the diverticular disease to involve the whole sigmoid and descending colon. Sigmoidoscopic examination on several occasions was negative. Her symptoms were controlled by medical management.

Physical examination on the morning of her admission on July 26, 1960, revealed an elderly, confused, extremely pale woman, passing bright red blood per anum. Her blood pressure on admission was 80/50 mm. Hg; her pulse was 68 per minute and irregular. Abdominal examination revealed slight distension; no enlargement of liver or spleen, and no masses or tenderness; the bowel sounds were hyperactive. Sigmoidoscopy (up to 14 cm.) revealed large amounts of fresh blood in the rectum and recent clots; no bleeding point could be visualized.



Fig. 1.—Opened sigmoid colon with orifices of three diverticula visible and a blood clot protruding through the orifice of the diverticulum on the left side.

Preoperative Course

Whole blood transfusions, vitamin K and calcium gluconate were given as soon as possible, but even after 10 units of blood she continued to pass an almost continuous stream of red blood and her blood pressure failed to rise.

A tentative diagnosis of massive hemorrhage from diverticular disease of the sigmoid colon was made and laparotomy was performed five hours after admission to hospital.

*From the Departments of Surgery and Pathology, McGill University and The Montreal General Hospital.



Fig. 2.—Section of the bowel wall through the diverticulum. The dark clot is visible in the lumen, the inflammatory induration about the diverticulum in the pericolic fat is apparent and the site of the disrupted vessel can be discerned (at arrow point).

Operation

Under general anesthesia, the abdomen was opened through a lengthy left paramedian incision. The colon and distal 20 cm. of terminal ileum were found to be distended with fresh blood, and numerous diverticula were found in the sigmoid and descending colon. Examination of the stomach, duodenum and small intestine was negative.

To rule out the possibility of a proximal source of bleeding in the colon, openings were made in the cecum and transverse colon and the bowel was examined through a sigmoidoscope. No lesion was seen. Following this, a left hemicolectomy was performed with end-to-end anastomosis. A transverse colostomy and cecostomy were performed, utilizing the previously made colotomy and cecotomy openings.

The patient made an uneventful recovery; the cecostomy closed spontaneously and the transverse colostomy was closed four weeks after the resection. She was discharged from hospital two months after admission with no evidence of recurrence of bleeding.

Pathological Findings

The fixed specimen measured 34 cm. in length. There was no gross evidence of inflammation. The ostia of seven diverticula could be seen, from one of which a thrombus was protruding.

In addition to this, three inverted diverticula were found in the specimen.

Attention was directed to the diverticulum containing the thrombus and this portion of the colon was sectioned in such a way as to bisect the diverticulum. A small circular zone of relative induration was seen in the wall of the diverticulum (Fig. 2), and the fat opposite this point was also noted to be indurated. There was no gross evidence of ulceration. Serial sections through this area eventually

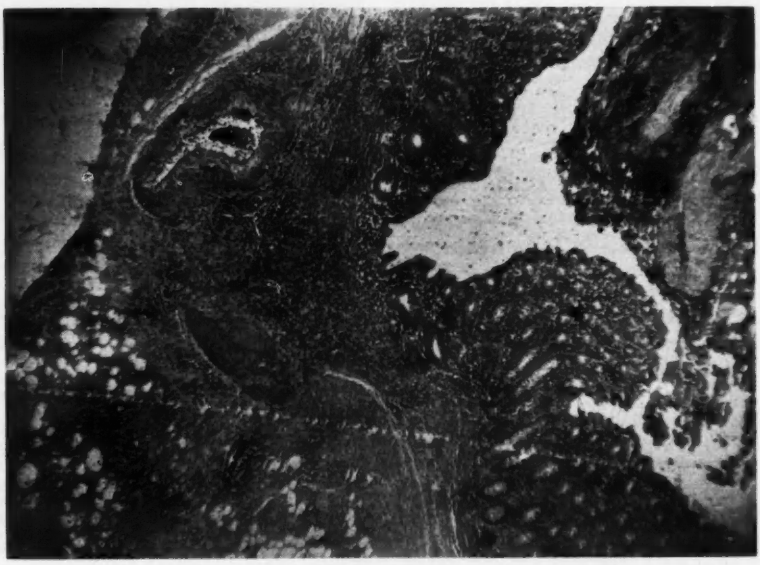


Fig. 3.—Microsection of diverticulum to show dark clot in the lumen at the right side, a re-epithelialized ulcerative crypt in the mucosal lining at the centre, and above this to the left side an artery incorporated in a peridiverticular inflammatory reaction of the pericolic fat.

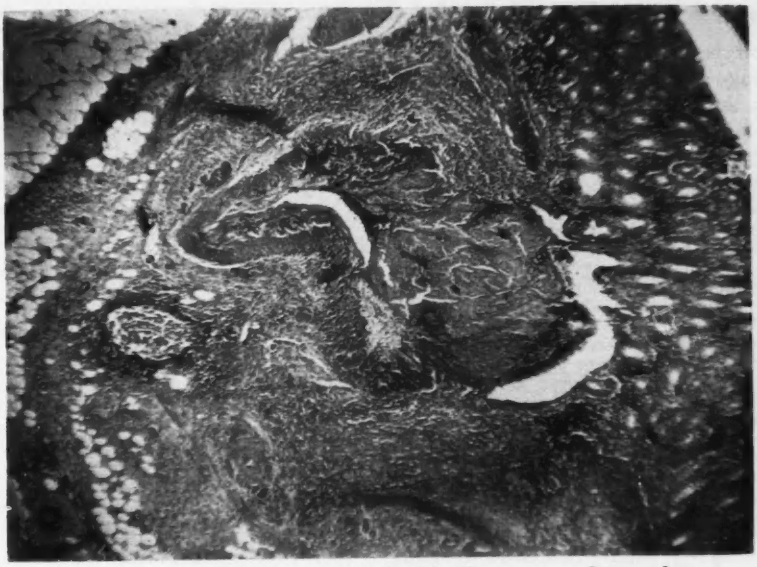


Fig. 4.—The artery is seen to the left of centre at a deeper plane. Its wall on the right side is deficient and a mass of clot rests in a tissue space between the defect and overlying mucosal tissue.

revealed (1) thrombus in the lumen of the diverticulum (Figs. 1, 2 and 3); (2) an area of healing ulceration of the mucosa (Fig. 3), and (3) a zone of inflammatory reaction in the bed of the ulcer and, traversing this zone of inflammatory reaction, a medium-sized artery with a defect in a segment of its wall filled with partially organized thrombus which extended well out into the perivascular tissues (Figs. 3, 4 and 5).



Fig. 5.—The eroded artery with intact wall to the left side; the total defect of the wall and the extruded clot to the right side.

Summary

A case is reported exhibiting massive hemorrhage from an eroded vessel in the wall of a diverticulum.

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Résumé

Ceci est l'histoire d'un cas d'hémorragie massive due à un diverticule du côlon.

Il s'agit d'une femme de 79 ans, admise à l'Hôpital Général de Montréal le 26 Juillet 1960. Deux semaines avant son admission, elle a présenté des saignements rectaux. Quatre années auparavant, elle avait été examinée dans le département des malades ambulants du même hôpital pour un syndrome de constipation alternant avec des pertes de sang et l'on avait établi déjà à ce moment une diverticulose du sigmoïde et du côlon descendant.

Elle reçut immédiatement les transfusions de sang que son état nécessitait: ceci eut pour résultat d'augmenter considérablement l'importance de l'hémorragie rectale. Une laparotomie exploratrice fut immédiatement décidée. On trouva un côlon distendu par le sang, porteur de nombreux diverticules. On pratiqua alors une hémicolectomie avec anastomose bout à bout. Une colostomie transverse et une cæcostomie de décharge furent faites.

Les suites opératoires furent excellentes. La malade put quitter l'hôpital deux mois après son admission.

Dans la pièce opératoire l'examen anatomopathologique permit de trouver un diverticule obstrué par un thrombus, au fond duquel une artériole se trouvait sectionnée par un ulcère.

L'intérêt de ce cas réside dans le fait qu'il a été possible de trouver avec exactitude le point responsable de l'hémorragie.

The *Canadian Journal of Surgery* is published quarterly by the Canadian Medical Association. Subscription rates are $10.00 a year or $2.50 for individual issues. Yearly subscriptions and back copies are available on request from the Canadian Journal of Surgery, C.M.A. House, 150 St. George Street, Toronto 5, Ontario.

RUPTURE AND STENOSIS OF MAIN STEM BRONCHUS

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STENOSIS of main stem bronchi after traumatic tears, other than those caused by endoscopy procedures, is now being recognized and treated with increasing frequency by bronchoplastic procedures or resection and anastomosis. The following is an illustrative case report.

CASE REPORT

A 17-year-old girl was brought to the emergency department of the Ottawa Civic Hospital at 11 p.m., on September 13, 1960. She had been in an automobile accident and had sustained a compression injury to her chest. The chest was compressed between the right front seat and the dashboard of the automobile in which she was riding. The patient complained of severe anterior chest pain on the left side and difficulty in breathing. She was coughing up bright red blood clots.

Examination revealed a teen-age girl not in acute distress. Her blood pressure was 80/60 mm. Hg, pulse rate 96 per minute and respirations 32 per minute. There were some very superficial abrasions on the left chest anteriorly. Apart from these there were no other marks. Auscultation of the chest revealed good air entry bilaterally with a few adventitious sounds on the left side of the chest. Chest radiographs at this time revealed a 5% to 10% pneumothorax on the left side and a lesser pneumothorax on the right side, without evidence of bony injury.

Within one hour the patient's condition improved. She did not have nearly as much pain; difficulty in respiration had been alleviated with oxygen, and her blood pressure was 110/90 mm. Hg.

The following morning on examination the patient again had difficulty with respirations. There was gross surgical emphysema bilaterally in the neck, extending down to, but not below, the clavicles. Her blood pressure was 90/80 mm. Hg and her respirations were 30 per minute. Laryngoscopy revealed a small tear just below the cricoid cartilage posteriorly. A chest radiograph revealed a 40% to 50% pneumothorax on the left side and no pneumothorax on the right (Fig. 1). Surgical consultation was obtained and underwater intrapleural drainage into the left chest was established.

By September 15 the pneumothorax on the left side had been reduced to 10% and the intrapleural drainage had ceased to function. Clinically, by September 17 there was good air entry into the left and right sides of the chest and the intrapleural drainage was removed. On September 19 a slight tachycardia which had been present since September 15 increased to 140 per minute and there was no air entry to the left chest (Fig. 2). Bronchoscopy was performed that day. The post-cricoid tear seen by the laryngoscopist was again noted and appeared to be healing. The left main stem bronchus was hyperemic, edematous and contained a large blood clot just proximal to the left upper lobe take-off. Aspiration of the left main stem bronchus was performed, after which the basilar segments of the lower lobe could be viewed easily and no other abnormality was noted in the left main stem bronchus.

There was considerable improvement of air entry to the left chest the following day but this was not as marked as expected. Radio-

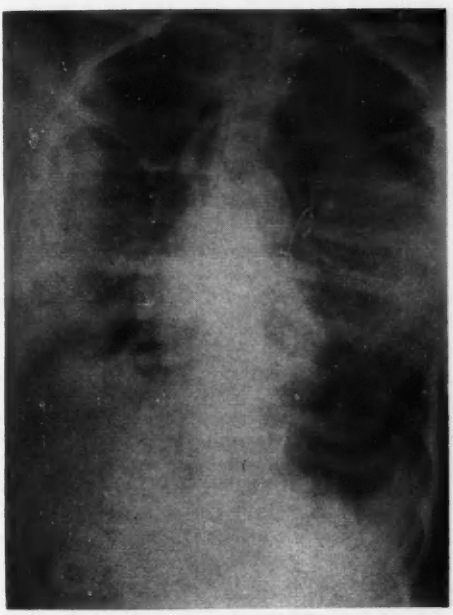


Fig. 1.—Pneumothorax—left side.

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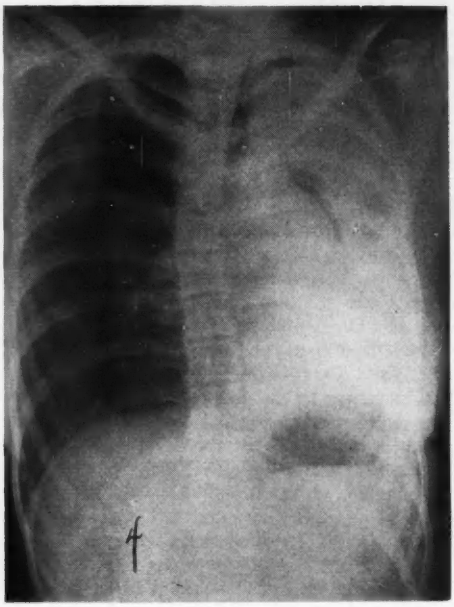


Fig. 2.—Atelectasis—left side.

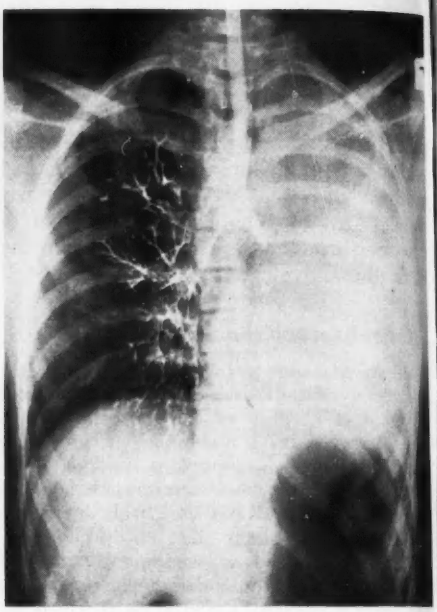


Fig. 3.—Bronchogram demonstrating complete occlusion of left main stem bronchus.

graphs taken on September 23 and September 26 revealed evidence of air entry to the left chest with a haziness over the left lower lobe. The patient during this interval had been using a Bird respirator with aerosol therapy. Sputum was minimal and was not blood-tinged. However, after September 26, air entry decreased and became absent, with complete atelectasis of the left lung during the next five days.

Bronchoscopy and bronchography performed on October 3 revealed complete obstruction of the left main stem bronchus proximal to the left upper lobe take-off (Fig. 3). The history was again reviewed with regard to the possibility that a foreign body might have been aspirated at the time of the accident, but no such information could be obtained.

On October 12 a left thoracotomy was performed using a posterior approach. There were minimal adhesions within the chest cavity and both upper and lower lobes were completely atelectatic. Dissection of the left main stem bronchus was difficult due to numerous adhesions between the main stem bronchus and the pulmonary artery. The pulmonary artery was isolated and an umbilical tape was placed around it in the event that it might be torn. At a level approximately 2 cm. proximal to the left upper lobe take-off there could be seen and felt a definite area of stenosis of the left main stem bronchus which extended up to and under the aortic arch. To mobilize the bronchus completely, the aorta was mobilized and the bronchus was then freed up to the carina. The bronchus was then severed just proximal to the left upper lobe take-off. The proximal bronchus was full of thick granulation tissue and no anesthetic gases could be forced through the severed end of the bronchus under direct pressure. Distal to this the lung contained thick tenacious gelatinous secretions, which required numerous washings to complete their removal. The bronchus was again severed 1 cm. distal to the carina at a level proximal to the stenosis. With considerable difficulty a primary anastomosis was carried out between the two ends of the left main stem bronchus using atraumatic 000 silk sutures. Both lobes of the lung then expanded without difficulty. Intrapleural drainage was established after the anastomosis had been covered with mediastinal pleura.

Immediately postoperatively there was good air entry and full expansion of both upper and lower lobes of the left chest. Satisfactory air entry was maintained until the evening of October 13, the first postoperative day, when there was marked decreased air entry into the

left chest and a radiograph confirmed a recurrence of atelectasis.

Bronchoscopy was performed on October 14 and very thick tenacious plugs were removed from the left main stem; the plugs extended well down below the suture line. The suture line appeared to be intact and the lumen of the main stem was approximately two-thirds of the normal diameter.

Subsequent to this the patient had an uneventful postoperative course. The chest drainage tube was removed on the fourth postoperative day, the sutures were removed on the eighth postoperative day and the patient was discharged on the fourteenth day. Radiographs on discharge revealed good aeration of the left chest (Fig. 4).

One month after the bronchial resection and anastomosis the patient was readmitted to hospital for bronchoscopy; this revealed a narrowed left main stem bronchus which nevertheless had an adequate-sized lumen without secretions. Chest radiographs at this time revealed good air entry into the left chest.

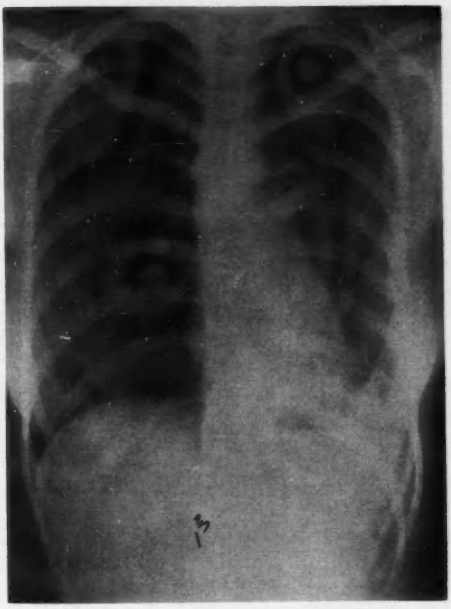


Fig. 4.—Complete aeration of left lung.

Discussion and Summary

The mechanism of injury in this case is difficult to establish; three possibilities have to be considered in an explanation: (1) forced expiration with a closed glottis causing positive intrathoracic pressure (Valsalva's phenomenon); (2) normal angulation of the left main stem bronchus compared with the right main stem bronchus with sudden deceleration producing a shearing force causing a tear in the bronchus, and (3) external compression to the chest.

This patient had a tear of the left main stem bronchus which progressed to complete bronchial stenosis. Such lesions usually do not present dramatically with the patient in critical condition, and usually there are no associated chest injuries such as rib fractures. It is interesting that the stenosis occurred within 15 days of the initial injury in this case and that by resection of the stenosed area and primary anastomosis it was possible to conserve all the healthy lung tissue in the left chest. This is certainly more satisfactory, both to the patient and to the surgeon, than a pneumonectomy.

The development of a stricture at the site of anastomosis may occur at a later date as has been reported by others.[1] Repeated dilatation by bronchoscopy may then be required to maintain an adequate-sized lumen of the left main stem bronchus. However, after an interval of five months this patient has returned to normal activity and is participating in athletic activities; thus it would seem improbable that a stricture will occur.

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Résumé

Ceci est l'histoire d'un cas.

Une jeune fille de 17 ans, victime d'un accident d'automobile, a eu le thorax comprimé entre le

tableau de bord d'une voiture et le dossier de son siège. Elle souffre, a de la difficulté à respirer et en toussant expectore des caillots de sang. Son état général est assez bon; la radiographie pulmonaire montre un léger pneumothorax bilatéral. Elle est placée sous une tente à oxygène et son état s'améliore rapidement.

Le lendemain, la difficulté à respirer revient. Une nouvelle radiographie montre une augmentation du pneumothorax à gauche; par contre, il y a eu résorbtion à droite. On pratique une bronchoscopie qui permet d'aspirer un gros caillot obstruant la bronche souche gauche; de plus, on installe une aspiration pleurale continue.

Une dizaine de jours plus tard, il apparait un syndrome d'atélectasie gauche. On pratique alors une thoracotomie postérieure. La dissection de la bronche souche gauche se fait difficilement à cause de nombreuses adhérences avec l'artère pulmonaire. Une sténose de cette bronche, située à 2 cm. de son entrée dans le parenchyme est découverte: elle est due à une masse de tissu de granulation. On pratique la résection de cette portion et on anastomose les deux extrémités avec difficulté par des sutures à la soie 000. Les suites opératoires furent bonnes et la malade put quitter l'hôpital deux semaines plus tard.

Elle a été revue depuis: à la bronchoscopie, il existe un rétrécissement partiel du canal bronchique, bien que celui-ci soit suffisamment large pour permettre une ventilation tout à fait satisfaisante.

THE TIBIALIS ANTERIOR SESAMOID

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Accessory ossicles of the foot lying in relation to the medial aspect of the first cuneiform and navicular are uncommon and there has been some confusion regarding their terminology. The purpose of this paper is to review the literature on this subject and to report a patient with a tibialis anterior sesamoid which was removed at operation.

Dwight,[2] in 1902, described an accessory ossicle lying medially in a hollow between the navicular and the first cuneiform. He suggested that the title "paracuneiforme" be used for anomalous ossicles occurring in relation to the first cuneiform, with the exception of the os intermetatarsium and the os intercuneiforme. Zimmer[5, 6] identified two types of os paracuneiforme: (a) a small ovoid bone lying in a groove between the navicular and the first cuneiform, and (b) the tibialis anterior sesamoid lying entirely medial to the first cuneiform. He described a tibialis anterior sesamoid which he dissected from the foot of an amputated specimen. O'Rahilly[4] clearly distinguished between the os paracuneiforme lying in relation to the navicular and first cuneiform, and the tibialis anterior sesamoid lying medial to the first cuneiform; it now appears preferable to accept these two terms as indicating distinct and separate entities.

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Carlier[1] and Morrison[3] have reported patients with anterior tibial sesamoids. Both of these patients presented with symptoms relating to the accessory bone, which at operation was found lying in the tendon of the tibialis anterior, articulating with the medial aspect of the first cuneiform.

Case Report

A six-year-old girl was seen in December 1959 with the chief complaints of pain and deformity of the left foot. On examination there was mild pronation of both feet. The tibialis anterior tendon of the left foot extended to a tender slightly movable bony hard prominence on the dorsomedial aspect of the first cuneiform. The tendon appeared to reach subcutaneously from this bony prominence to the region of the medial aspect of the base of the proximal phalanx of the great toe. A small accessory toenail was present posteromedial to the nail of the great toe (Fig. 1). A mild hallux adductus was present, and on active dorsiflexion of the foot there was a cock-up deformity of the great toe. Radiological studies of the left foot revealed two ossicles contiguous to the medial aspect of the first cuneiform, and a small calcific-like density medial to the base of the distal phalanx of the great toe, adjacent to the site of the accessory toenail (Fig. 2). Operation was performed in June 1960. The sesamoid, in two parts, was dissected out of the tibialis anterior tendon. The deep portion articulated with a facet on

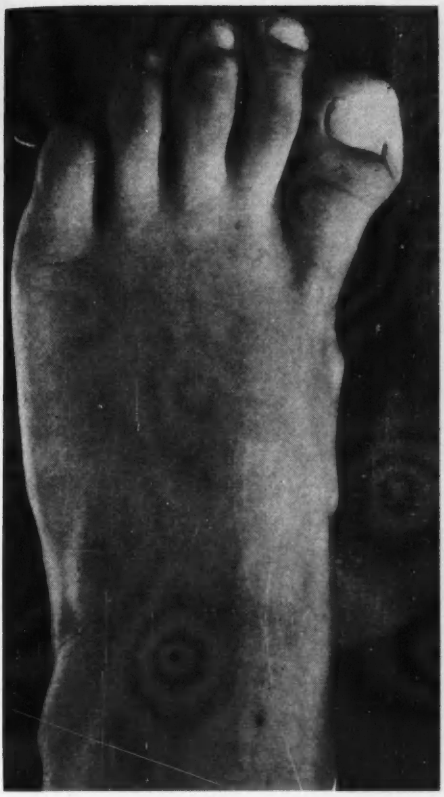


Fig. 1



Fig. 2

Fig. 1.—Photograph of the dorsum of the patient's foot showing: (a) the prominence of the sesamoid tibialis anterior medial and proximal; (b) puckered skin owing to anomalous tendon insertion, and (c) accessory toenail on short great toe. **Fig. 2.**—Postero-anterior radiograph of foot reveals: (a) bi-partite accessory ossicle adjacent to the medial proximal aspect of the medial cuneiform; (b) a muscular-like band extending from the accessory ossicle to the region of the base of the proximal phalanx; (c) a 2-mm.-long density medial to the base of the distal phalanx at the base of the accessory toenail, and (d) a slightly shortened first metatarsal and shortened phalanges of the great toe.

the medial surface of the first cuneiform. At the site of the sesamoid the tibialis anterior tendon expanded into three bands presenting a cruciate appearance. One band was inserted into the dorsum of the first cuneiform and another inserted into the skin on the medial aspect of the first cuneiform; the third extended distally inserting into the periosteum of the medial aspect of the distal portion of the first metatarsal and diffusely into the overlying puckered skin. The accessory nail and the distal portion of the tendon were excised, and the remainder of the tendon was sutured to the periosteum on the medial aspect of the base of the first metatarsal.

Discussion

Accessory bones of the foot are usually found as incidental radiographic findings, often of no clinical significance. The patient described was an exception in that there was pain related to the sesamoid, the foot was deformed and there was a mild disturbance of muscle balance of the foot associated with the anomalous insertion of the tibialis anterior tendon. Operation was undertaken to relieve pain and to improve the appearance of the foot.

The occurrence of the sesamoid, the aberrant attachment of the tibialis anterior

tendon and the accessory toenail with an adjacent ossific centre might be interpreted as a primitive attempt at the formation of an extra ray of the foot.

Summary

The literature on accessory ossicles of the foot related to the medial aspect of the navicular and first cuneiform has been reviewed.

The case report of a patient with a tibialis anterior sesamoid removed at operation is presented.

The case presented is unusual in that the sesamoid was bi-partite and was associated with an anomalous attachment of the tibialis anterior tendon and an accessory great toenail.

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Résumé

Résumé d'une histoire de cas. Il s'agit d'une petite fille de six ans vue en Décembre 1959, se plaignant de douleurs dans le pied gauche. L'examen révèle une légère pronation des deux pieds et l'on peut palper dans la partie inférieure du tendon tibial antérieur une petite tumeur dure. A la radiographie on découvre de petits os surnuméraires: deux à proximité du premier cunéiforme et un troisième en dedans de la phalangine du gros orteil. Ces formations furent enlevées par opération en Juin 1960.

Après avoir passé en revue la littérature sur le sujet, les auteurs notent que la présence d'os sésamoïdes dans le pied est généralement sans symptomatologie. Souvent, ce n'est qu'une trouvaille radiologique. Dans le cas présenté ici, la malade accusait des douleurs; de plus il existait une déformation du pied et l'insertion du tendon du muscle tibial antérieur se faisait de façon anormale.

CLINICAL NEUROSURGERY. Proceedings of the Congress of Neurological Surgeons, Miami Beach, Florida, 1959. Vol. 7. 278 pp. Illust. The Williams & Wilkins Company, Baltimore, Md., 1961. $12.00.

This seventh volume of "Clinical Neurosurgery" is comprised of the edited proceedings of the ninth annual meeting of the Congress of Neurological Surgeons held in October 1959. The volume is dedicated to Dr. William J. German, professor of neurological surgery at Yale University School of Medicine. It contains excellent papers by other neurologists and neurosurgeons which are related mainly to brain tumours, either from the diagnostic or therapeutic point of view.

The contributions by Dr. German include a follow-up study of gliomas and acoustic neuromas. These are thoughtful essays and are concerned with many of the patients originally operated upon by Dr. Harvey Cushing.

Other clinical papers include considerations of metastatic tumours of the brain, tumours involving the third ventricle, surgical techniques for removal of intracranial meningiomas, epilepsy and brain tumour, isotope localization of brain tumours and the results of radiotherapy. Disorders of visual perception in patients with brain tumours and an argument for the small pneumoencephalogram represent other chapters.

More basic studies include a paper on electron microscopy of brain tumours, observations on gliomas transferred from guinea-pigs' eyes to cell cultures, and a report on the results with chemotherapy of gliomas by vascular perfusion of antitumour agents. The final chapter by Dr. Zimmerman discusses the nature of experimental gliomas.

This book has a rich variety of contributions by an impressive list of experts. It will be found invaluable to neurologists and neurosurgeons and other specialists in nervous diseases. Some, but by no means all, of the papers will be of more general interest.

EXPERIMENTAL SURGERY

FURTHER EXPERIENCES WITH THE USE OF NITROGEN MUSTARD AS AN ADJUNCT TO OPERATION IN THE TREATMENT OF CANCER*

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NITROGEN mustard was given to patients at the time of operation for cancer to determine if its use was safe and if it decreased the incidence of tumour recurrences. The original procedure has been outlined previously.[1] In patients with intra-abdominal growths the drug was placed in the lumen of the gut, operative wounds and peritoneal cavity; in patients with growths elsewhere nitrogen mustard was given by the systemic venous route and operation wounds were irrigated with a solution containing the drug.

Recently there has been increasing interest in both the physical and chemical measures that may be taken at the time of operation for cancer in an attempt to decrease the incidence of local and distant metastases. Physical procedures that have been advocated for gastrointestinal tumours are minimal handling, early ligation of blood supply, the placing of tapes above and below the growth to occlude the lumen of the gut, and the covering of the peritoneal surface to prevent the spread of cells from the serous surface. A growth situated outside the abdomen should be handled as little as possible both before and during operation, and the blood supply to the region should be divided at an early stage during operation. Many chemicals, such as saline, Dakin's solution and Clorpactin XCB, have been used in operative wounds in an attempt to prevent local recurrences. The lumen of the gut has been washed out with perchloride of mercury[2] and Clorpactin XCB[3] to prevent implants occurring at the site of intestinal anastomosis. Attempts have also been made to control cancer cells in the blood stream and cells which are already established as early metastases by administering cytotoxic agents, such as nitrogen mustard (Mustargen) and triethylene thiophosphoramide (Thio-TEPA).[4] Efforts have been made to increase the general resistance of the patient by raising the properdin level in the blood,[5] and various antisera[6] have been prepared to act directly on the cancer cells.

Nitrogen mustard (methyl-bis [β-chloroethyl] amine hydrochloride; HN_2; Mustargen; Mechlorethamine Hydrochloride N.F.) has been studied most intensively as a cytotoxic and cancericidal agent. It belongs to the group of polyfunctioning alkylating agents which give rise to a highly active pentavalent nitrogen atom and a chloride anion. The rate of inactivation of the nitrogen atom depends on the pH, temperature and the amount of organic material present in the medium. The hydrogen ion concentration of a solution of nitrogen mustard, freshly prepared with unbuffered saline, is 4.8 pH units; this falls rapidly to a pH of 3.8 during the first hour and then more slowly to a steady level of about 3.4 pH units in 24 hours. During the initial acidification the parent amine molecule is undergoing a transformation into the imine form at a rate corresponding to a half-life of 90 seconds.[7] The imine molecule is more stable in aqueous acidic solution than the parent amine and remains both toxic and cancericidal to animals after standing for 24 hours at room temperature (25° C.).[8] When introduced into the body (pH 7.3) the imine and remaining amine molecules react rapidly with a large number of organic materials, particularly nucleoproteins, and probably remain biologically active for only a few minutes. The reaction within the body is non-specific, affecting mostly cells which are undergoing rapid mitosis such as those

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Presented at the Southwestern Ontario Regional Meeting, the Royal College of Physicians and Surgeons of Canada, London, Ont., November 15, 1960.
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in the bone marrow, intestinal mucosa, testis and malignant tumours. Since chromosome damage, systemic toxicity and therapeutic response are in many ways similar to those following X and gamma radiation, the drug is often described as radiomimetic.

Nitrogen mustard has been used extensively for the treatment of established malignant disease. Some of the neoplastic conditions of the lymphoid and hemopoietic systems, e.g. Hodgkin's disease, are controlled for short periods of time, whereas the response of carcinomata and sarcomata depends upon the amount of the drug which can be delivered to the tumour. When nitrogen mustard is given by the systemic venous route and passes through the pulmonary capillary bed before arriving at the growth, there is generally no decrease in the size of a tumour and only occasionally symptomatic relief. However, when the drug is given intravenously and reaches the tumour without first passing through a capillary bed (as with primary and secondary lung neoplasms), the growth may become smaller and there is frequently relief of symptoms. The intra-arterial injection of the tumour-bearing area on one or more occasions may further enhance the effect of the drug.[9] Creech *et al.*[10] found that an even greater dose of nitrogen mustard could be delivered by isolating the tumour-bearing region from the general circulation and perfusing it through an extracorporeal circuit. The most suitable sites for this procedure are the limbs, although it has been used for growths in the pelvis, lungs, breasts and brain. Damage to blood vessels limits the dose to the limbs, whereas at other sites the dose is limited by leakage from the perfusion circuit into the general systemic circulation. Direct instillation of the drug into the appropriate body cavity often causes a decrease in effusions due to pleural or peritoneal metastases.

As an adjunct to surgery, nitrogen mustard has only recently been used in an attempt to decrease local and distant metastases. Mrazek *et al.*[11] gave the drug to patients with gastrointestinal cancer at the time of operation by the intraportal and intraperitoneal routes. One-tenth of a milligram was given into a radicle of the portal vein and the same dose in 50 ml. of physiologic saline into the peritoneal cavity. In patients suffering from carcinoma of the breast, 0.2 mg./kg. body weight of the drug was injected into a systemic vein at the time of operation. Both groups received 0.1 mg./kg. body weight by the systemic venous route on the two days following operation. Preliminary results suggested that the number of cancer recurrences was less than in untreated patients and that serious complications did not follow the use of the drug. The most marked effect was a decrease in the white blood cell count to an average of 3900 cells/mm.^{3} on about the twelfth day. Wound infection and impairment of wound healing were not a problem, although when they occurred in association with leukopenia they tended to be more severe. The effect of the drug on the hemoglobin concentration was less than that on the white blood cell count. Large doses of nitrogen mustard have been given into the peritoneal cavity at the time of operation without the development of complications; Morales *et al.*[12] state that Curreri gave 22 mg. and 30 mg. to two patients and Mengert gave 30 mg. to four patients. Operative wounds have been irrigated with a solution containing nitrogen mustard in an attempt to decrease the incidence of cancer recurrences; Kredel[13] used 10 mg. in 500 ml. of physiologic saline for this purpose; he found that healing was not impaired and that skin grafts could be applied safely.

The toxic effects and cancericidal properties of nitrogen mustard have been extensively investigated in animals. When applied directly to wounds in rats[14] the drug does not impair healing when a concentration of 2.5 mg./100 ml. of physiologic saline is used. The systemic venous administration of the drug to rats,[14] rabbits[15] and dogs[16] does not interfere with wound healing unless a dose is given which kills a number of the animals. Cruz, McDonald and Cole[17] found that the injection of nitrogen mustard (0.5 mg./kg. body weight) into the portal venous system of rats one minute after giving 10,000 Walker 256 tumour cells by the same route caused a decrease in the number of tumours developing in the liver. Injection of the drug more

than one hour before or six hours after injection of the cells did not have much protective effect. The most effective route for the administration of the drug and its effect on cancer cells implanted at various sites have been studied.[18] Nitrogen mustard injected intraportally, intraperitoneally or by a systemic vein is equally effective in reducing the number of liver tumours after injection of Walker 256 tumour cells into the portal venous system. The drug is therefore absorbed in an active form into the portal circulation from the peritoneal cavity. When Walker 256 cells are introduced into the abdominal cavity of rats, the number of animals which develop peritoneal implants is less following the intraperitoneal injection of the drug than after its intraportal or systemic venous administration. These results suggest that in patients suffering from gastrointestinal cancer, nitrogen mustard may be most effective in reducing the number of peritoneal and liver metastases when given into the peritoneal cavity, rather than by a combination of the intraportal, intraperitoneal and systemic venous routes. Nitrogen mustard is a potent cancericidal drug when directly applied to cells of the Walker 256 tumour; when 10,000 Walker tumour cells were implanted into subcutaneous pockets on the abdomens of rats and one hour later the wounds were irrigated with 100 ml. of a solution containing 1 mg. of nitrogen mustard in 100 ml. of physiologic saline, the number of tumours developing at the operative site was reduced from 89% to 3%.[19]

Materials and Methods

In patients with gastrointestinal cancer, the lumen of the gut was occluded with tapes above and below the tumour, and the blood supply was divided before the growth was mobilized. When the growth was in the upper rectum and sigmoid colon it was not always possible to apply a tape below the tumour. A solution containing 10 mg. of nitrogen mustard (Mustargen) in 500 ml. of physiologic saline was injected into the lumen of the gut proximal and distal to the tapes and removed after resection of the tumour. A second solution of nitrogen mustard (0.3 mg./kg. body weight at a concentration of 2.5 mg./100 ml. of physiologic saline) was placed in the operative wound and abdominal cavity just before closing the peritoneum, and left in the abdomen. After abdominoperineal resection of the rectum, the perineal wound was irrigated with 10 mg. of nitrogen mustard in 500 ml. of physiologic saline. In patients suffering from cancer at sites outside the abdominal cavity, e.g. breast, cervical glands, lung and kidney, the operative site was irrigated with 10 mg. of the drug in 500 ml. of physiologic saline, and after removal of the tumour, nitrogen mustard (0.3 mg./kg. body weight in 20 ml. saline) was injected into the median basilic vein. The dose of the drug and volume of fluid used were calculated from a graph (Fig. 1). The hemoglobin concentration, white cell and platelet counts were determined before operation and on the first, fourth, seventh, tenth, fourteenth, seventeenth and twenty-first postoperative days; in 10 patients more frequent observations were made. The volume of gastric suction, drainage from operation sites, wound healing, period of hospitalization and the occurrence of other complications were recorded.

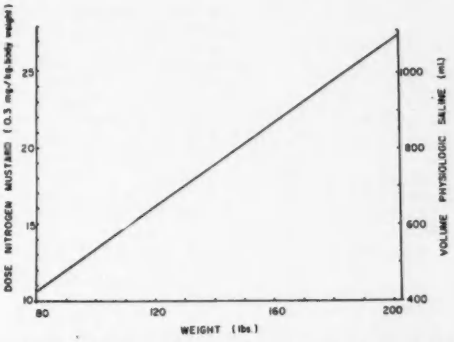


Fig. 1.—Dose of HN_2 (0.3 mg./kg. body weight) and volume of physiologic saline (2.5 mg. HN_2/100 ml.) used in patients at the time of operation.

Results

Thirty-two patients with intra-abdominal cancer (Table I) received nitrogen mustard in the lumen of the gut, peritoneal cavity and operative wound; after abdominoperineal resection of the rectum, five had the perineal wound irrigated with a

TABLE I.—Clinical Observations in Patients Receiving Nitrogen Mustard by the Intraperitoneal Route and at the Operation site

Site	Number of cases	Mean number (±S.D.) of days of gastric suction	Mean number (±S.D.) of days in hospital	Wound infection	Other complications
Stomach	2	3.0	30	0	0
Hepato-biliary and pancreas	2	4.0	32	0	0
Colon:					
Treated	23	3.6 ± 1.4	17.3 ± 2.8	0	1 intestinal obstruction 1 bronchopneumonia
Controls	10	3.6 ± 1.3	29.7 ± 14.5	1	1 pulmonary embolism 1 intestinal obstruction 1 general peritonitis
Rectum:					
Treated	5	3.1 ± 1.2	41.8 ± 20.7	3	0
Controls	8	3.1 ± 1.0	29.2 ± 13.7	0	1 bronchopneumonia

solution containing the drug. In the 23 patients with cancer of the colon there was no increase in the number of days of gastric aspiration (4 days), and the average period of hospitalization (17 days) was not increased. However, in patients who had abdominoperineal removal of the rectum, the average period in hospital was increased (42 days) due to the fact that three of the five perineal wounds became infected. Patients who received the drug only locally and in the peritoneal cavity did not have greater lethargy and anorexia. Rotation of the gut at the site of intestinal anastomosis caused intestinal obstruction in one patient after removal of a carcinoma of the pelvic colon; at the second operation ten days after the use of the drug there was no increase in peritoneal reaction. The white blood cell count (Fig. 2) increased

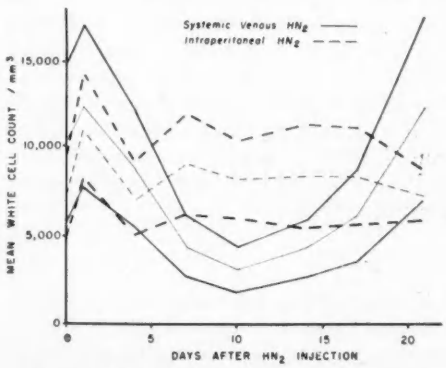


Fig. 2.—White blood cell count in patients receiving HN_2 (0.3 mg./kg. body weight) at the time of operation (1 S.D. plotted above and below the mean).

to 11,300 cells/mm.^{3} on the day after operation and returned to a normal level on the fourth day. The hemoglobin concentration decreased slightly; a total of 5600 ml. of blood was given to six of the 32 patients following operation. There was no change in the platelet count. In five patients who had detailed blood studies performed after operation, leukocytosis was found to occur on the first to third days and to return to the normal value by the fourth day. The lowest white blood cell count recorded was 4400 cells/mm.^{3}

Twenty-seven patients with growths outside the abdominal cavity (Table II) had their operative wounds irrigated with nitrogen mustard (2.0 mg./100 ml. of physiologic saline), and nitrogen mustard (0.3 mg./kg. body weight) was injected into a systemic vein at the time of operation. In 18 patients who had radical mastectomy performed for carcinoma of the breast there was a slight increase in the number of days of wound suction (5 days, controls–4 days) and the volume of fluid was increased (447 ml., controls–257 ml.). The average time spent in hospital was increased from 20 to 29 days because of the occurrence of wound infection in six patients. Extensive wound infection and empyema occurred in one of the five patients with carcinoma of the lung. Lethargy and anorexia were encountered in many of the patients who received nitrogen mustard by the systemic venous route. The white blood cell count increased to 12,400 cells/mm.^{3} on the day after operation followed by a decrease to 3100 cells on the tenth day; it

TABLE II.—Clinical Observations in Patients Receiving Nitrogen Mustard by the Systemic Venous Route and at Operation Site*

Site	Number of cases	Mean number (±S.D.) of days of wound aspiration	Mean total volume (±S.D.) of aspirate from wounds (ml.)	Mean number (±S.D.) of days in hospital	Wound infection	Other complications
Breast:						
Treated	18	5.4 ± 3.5	447 ± 390	28.5 ± 16.4	6	0
Controls	34	4.5 ± 2.2	257 ± 226	19.7 ± 10.6	1	1 edema of arm.
Lung	5	3.0	517	35	1	1 empyema
Kidney	2	3.0	125	18	0	1 death on second day from pulmonary aspiration
Bladder	1	3.0	2340	12	0	1 death from pulmonary embolism
Lymph nodes	1	4.0	185	12	0	0

*(The control patients were treated by the same surgeons during the year preceding the use of nitrogen mustard; they are therefore not exactly comparable.)

then returned to the initial value within three to four weeks. There was a slight decrease in the hemoglobin value; 5800 ml. of blood was given to six of the 27 patients following operation. No change was observed in the platelet values. Detailed blood investigations carried out in five patients showed that leukocytosis lasted from the first to the third day after operation and leukopenia from the fifth to the fourteenth days. The lowest white blood cell count was 1100 cells/mm.[3]

Discussion

Because of the increased incidence of wound infections occurring in patients who were given nitrogen mustard by the systemic venous and local routes, the drug is now being given only in a single dose (0.3 mg./kg. body weight) by a systemic vein at the time of operation. The systemic venous dose is not being increased, because in order to have an anticancer action it is probably necessary for the drug to cause toxemia and leukopenia. Since the local use of nitrogen mustard in patients who received the drug by the systemic venous route caused impairment of wound healing and may have contributed to perineal wound infection, local application of the drug to wounds has been discontinued. A modified procedure is now being used and a random selection of treated and nontreated patients is being studied to determine the incidence of metastases. However, even by applying statistical methods of prediction, results will not be available for at least two years. In contrast to the experience reported here, McClure and Mengert[20] found a very high incidence of complications following the intraperitoneal administration of nitrogen mustard and recommended that its use should not be continued. This was probably because these investigators used both a higher dose and a higher concentration of nitrogen mustard.

A number of patients, for example those with cancer of the breast, received radiotherapy following the use of nitrogen mustard at the time of operation. The time of commencing radiotherapy was not delayed and the total dose of radiation was not reduced except in the small number of patients in whom there was impairment of wound healing. Nevertheless, the white blood cell count should be determined before giving radiotherapy and repeated observations should be made during treatment.

Summary

Nitrogen mustard was given to patients at the time of operation for cancer to determine if its use was safe and if it decreased the incidence of tumour recurrences. Thirty-two patients with intra-abdominal cancer received 2 mg. of nitrogen mustard in 100 ml. physiologic saline

into the lumen of the gut and a similar solution into perineal wounds after abdominoperineal excision of the rectum. Nitrogen mustard (0.3 mg./kg. body weight at a concentration of 2.5 mg./100 ml. of physiologic saline) was placed in the peritoneal cavity and operative wounds. Leukopenia, general toxicity, paralytic ileus and intestinal symptoms did not occur. However, there was impairment of wound healing in perineal wounds irrigated with the drug. It is suggested that the drug can be used safely in the lumen of the gut and peritoneal cavity and that its use should be discontinued in operative wounds. Twenty-seven patients with cancer outside the abdominal cavity received nitrogen mustard (0.3 mg./kg.) by a systemic vein at the time of operation and had their operative wounds irrigated with a solution containing the drug (2.0 mg./100 ml. of physiologic saline). Leukopenia and general toxicity occurred in these patients. There was in addition an increased incidence of wound infection; the drug, therefore, is now being given by the systemic venous route and is not being used directly in operative wounds.

ACKNOWLEDGMENTS

We would like to thank Dr. I. H. Smith, Director, London Clinic, and the Ontario Cancer Foundation, and Dr. A. D. McLachlin, Professor of Surgery, University of Western Ontario, for their encouragement in this work.

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RÉSUMÉ

La moutarde azotée fut administrée à des malades opérés pour cancer, lors de l'opération, dans le but de déterminer si son usage était sans danger et s'il réduisait la fréquence des récidives. Trente-deux patients reçurent ainsi 2 mg. de moutarde azotée diluée dans 100 ml. de solution physiologique, dans des cas de résection abdomino-périnéale du rectum: la drogue fut donnée localement, sous forme d'irrigation dans la lumière intestinale et sous forme d'injection dans la plaie périnéale. En outre, une certaine quantité (0.3 mg./kg. de poids en une solution de 2.5 mg./100 ml. de sérum physiologique) fut placée dans la cavité péritonéale et dans la plaie opératoire.

On n'observa ni leucopénie, ni intoxication générale ni aucun symptôme intestinal ou iléiforme. Cependant, des défauts de cicatrisation, des désunions se firent au niveau de la plaie opératoire. C'est pour cette raison que les auteurs suggèrent de continuer l'emploi de la drogue en irrigations intra-intestinales et intra-péritonéales, mais de supprimer les injections dans les plaies opératoires.

Dans des cas de cancer extra-abdominaux, 27 malades furent étudiés. On leur avait administré 0.3 mg./kg. de poids de moutarde azotée par voie intra-veineuse et leurs plaies opératoires avaient été irriguées avec une solution physiologique contenant 2.0 mg./100 ml. de la même drogue. Dans ces cas, on vit apparaître une leucopénie et des phénomènes d'intoxication générale; il y eut de plus une augmentation de la fréquence des infections de plaies. Depuis, la moutarde n'est plus administrée que par voie intra-veineuse.

L'ensemble de ces résultats est compilé ici sous forme de tableaux.

SURGERY OF THE ACUTE ABDOMEN. John A. Shepherd, Consultant General Surgeon, Liverpool Region. Formerly Lecturer in Surgery, University of Liverpool. Surgeon Captain R.N.R. Foreword by Sir Zachary Cope. 1228 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1960. $17.00.

This book is intended to be a reference text on surgery of the acute abdomen. Since there are so few texts of this nature, it helps to fill a large hiatus in medical literature. The foreword is written by Sir Zachary Cope, who has contributed generously to this field. The material recorded is drawn largely from the experience of the author, supplemented by a review of the pertinent literature.

The initial chapters deal with the principles of diagnosis and management of acute abdominal conditions. Thereafter chapter headings are anatomically arranged to include the parieties and retroperitoneum, as well as the intra-abdominal viscera. In the case of certain large categories, special chapters are set aside to discuss one particular problem; for example, perforated peptic ulcer. Within a chapter each condition is discussed as to history, incidence, etiology and pathology, signs and symptoms, treatment and prognosis. Discussion of abdominal pain patterns, acute abdominal conditions in the newborn and abdominal emergencies in time of war have been purposely omitted.

This book will have its greatest value as a ready reference on acute abdominal conditions. It should prove invaluable to the surgical trainee and the young surgeon in creating for them a perspective in acute abdominal conditions. There are too few such works available.

A possible way in which this work could have been improved would have been through the contributions of multiple authors. Nevertheless, it is rather remarkable that one surgeon should have had so wide and varied experience of acute abdominal emergencies.

TRAUMATIC LESIONS OF PERIPHERAL VESSELS. Carl W. Hughes, Lt.-Colonel, U.S. Army Medical Corps; Assistant Chief, Department of Surgery; Chief, General Surgery Service, Tripler U.S. Army Hospital, and Warner F. Bowers, Colonel, U.S. Army Medical Corps; Chief, Department of Surgery, Tripler U.S. Army Hospital, Honolulu, Hawaii. 197 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $8.00.

Lieutenant-Colonel Hughes and Colonel Bowers have come up with a timely and authoritative compendium of first-rate knowledge, based on a wide experience of all pertinent data relative to the traumatized peripheral vessel. The presentation is a most readable one, traversing in just under 200 pages, the historical, research, developmental and practical aspects of the specialized field of vascular surgery, particularly in their relation to injury. The authors show throughout the book that they are surgeons of the whole being, but with a dedicated interest in the particular aspect under consideration. Old taboos in vascular surgery are rightly dismissed and a full plea for adoption of improved procedures and techniques is repeatedly made. All areas of major trauma are fully discussed with detailed considerations of techniques and management and with emphasis on systemic as well as on regional factors. Selection of the proper timing and material of the reparative procedure of choice in each instance of arterial or venous injury, with full regard to their concomitant complications and sequelae, makes this tidy presentation a delight to one versed in vascular techniques and an essential to any surgeon dealing in trauma.

Nostalgia is seldom a factor in reviewing a surgical treatise, but the reviewer well remembers meeting Colonel Bowers at Tripler in Honolulu, and can fully understand his co-authorship of a book of real merit in the field of traumatic lesions of the peripheral vessels.

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DR. JOHN ALEXANDER GUNN



Canadian Pacific Railway Co.

Dr. John A. Gunn

Death brought to an end the distinguished career of Dr. John Alexander Gunn of Winnipeg, on August 8, 1960.

He was born in Stonewall, Man., in 1878, where his father, who had arrived in this country from Scotland in his early youth, headed the firm of John Gunn and Sons, railway contractors.

Dr. Gunn qualified for his Arts degree at Manitoba College in 1897 and received his degree in medicine from Manitoba Medical College in 1904. In 1907 he became medical superintendent of the Winnipeg General Hospital and later practised in partnership with Dr. R. J. Blanchard, one of Winnipeg's outstanding surgeons. He also did postgraduate work in surgery in the U.S.A. and Europe.

At the outbreak of war in 1914 Dr. Gunn went overseas as officer commanding No. 1 Canadian General Hospital and later became consulting surgeon to the Canadian Army. He was twice mentioned in despatches, and for his service in the field was awarded the O.B.E. and C.B. He was later awarded the honour of honorary surgeon to the King.

He returned to practise in Winnipeg after the war and became chief surgeon of the Winnipeg General Hospital and of St. Boniface Hospital. He was appointed professor of surgery, University of Manitoba, in 1934, and on his retirement in 1939 was appointed emeritus professor of surgery. He was chief of surgery at Deer Lodge D.V.A. Hospital from 1920 until 1945 and honorary consultant in surgery from 1945 until 1948.

During all this time Dr. Gunn's interests in professional and community affairs were many and varied. He was president of the Canadian Club of Winnipeg and of the Manitoba Club. He was also president of the Manitoba Division of the Canadian Medical Association and a vice-president of the Canadan Medical Association. He was a founding member of the American College of Surgeons and a charter member of the surgical division of the Royal College of Surgeons of Canada, serving as vice-president of the College and as a member of Council. He was also a charter member of the Canadian Association of Clinical Surgeons. Dr. Gunn was one of the organizers of the Winnipeg Corps of Commissionaires, and also served as Chief Medical Officer of the Canadian Pacific Railway, Manitoba District, from 1940 until 1947.

His keen and sincere interest in all of these activities resulted in his making innumerable friends both in professional circles and in many other branches of community life, not only in Winnipeg, but in many other centres in Canada and the United States. A Winnipeg visitor at any surgical centre on this continent was always met with the question "How is John Gunn?" and was asked to convey friendly and sincere greetings. As a surgeon, Dr. Gunn's reputation was widespread and the stories of his manual dexterity and speed of operating are legion. Indeed, few surgeons of his era or even today could equal his technical ability. When operating, he seemed to know exactly what to do and the surgical procedure was always performed in the shortest possible time and with infinitely delicate skill. To work with him as a surgeon and to know him as a man was a continual source of pleasure.

Although he did not make any show of it, Dr. Gunn was possessed of deep religious convictions which pervaded his daily life and influenced all his daily activities.

Dr. Gunn continued in active surgical practice until his retirement in 1953 at the age of 75. Even after that time, many of his old patients sought his advice and received it ungrudgingly as long as his health permitted.

He died in Deer Lodge Hospital, where he had performed surgical operations for so many years. His passing is mourned by countless friends in all branches of business and professional life in Winnipeg and elsewhere, and especially by his widow, who gave him constant and devoted attention during his last illness, and his two sons, who are in business in Winnipeg and who may well be proud of a father such as theirs. T.E.H.

NOTICES

THE ALEXANDER GIBSON MEMORIAL FUND

The University of Manitoba has established a fund to honour the memory of the late Alexander Gibson, M.D. The purpose of the fund is to provide an annual lecture by an outstanding authority on surgery or research as a tribute to the great contribution which Dr. Gibson made to the teaching and practice of medicine during his lifetime.

The inaugural lecture was given by Sir Walter Mercer, emeritus professor of orthopedics, University of Edinburgh, at the Manitoba Medical College, Winnipeg, in January 1961.

Dr. Alexander Gibson was born in Scotland and received his medical education at the University of Edinburgh. He was a brilliant student, winning every honour and prize available to him, an unequalled record at Edinburgh.

In 1913, at the age of 32, Dr. Gibson came to Winnipeg to be Professor of Anatomy at the Medical College of the University. During the First World War he saw service in India and Egypt, and his transport was torpedoed in the Adriatic.

In 1918, Dr. Gibson returned to Winnipeg and began his practice in orthopedic surgery. He was actively engaged in this type of work until his death in 1956 at the age of 73.

Dr. Gibson's several important original contributions and his 77 publications earned for him a world-wide reputation in orthopedic surgery. Foremost amongst these was his development of a new operation upon the hip which brought relief to sufferers from arthritis.

During his lifetime he received many honours. For his research work in anatomy, he was made Fellow of the Royal Society of Edinburgh. In 1920 he became a Fellow of the American College of Surgeons; later he was made a senior member of the American Orthopedic Society. He served as President of the Canadian Orthopedic Association and of the Winnipeg Medical Legal Society. He was a life member of the Winnipeg Medical Society and of the Scientific Club of Winnipeg, and a Senior Member of the Canadian Medical Association.

His teaching and hospital appointments were professor of anatomy, lecturer in applied anatomy, associate professor of surgery in charge of orthopedic surgery, University of Manitoba; orthopedic surgeon and consultant to the Winnipeg General Hospital, and director of orthopedic surgery at Deer Lodge Hospital.

In the Second World War he was in charge of a Canadian Red Cross Unit at Hairmyres, Lanarkshire, Scotland.

When the Royal College of Physicians and Surgeons of Canada met at Winnipeg in 1954, he delivered the Annual Lecture in Surgery.

It is felt that many of his former friends, colleagues and students may wish to contribute to the Alexander Gibson Memorial Fund. Cheques should be made out to the University of Manitoba, earmarked for the Alexander Gibson Memorial Fund, and mailed to the Comptroller of the University.

NOTICE FROM THE ROYAL COLLEGE OF SURGEONS OF ENGLAND

The Council of the Royal College of Surgeons of England has established a post of Adviser in Surgical Training, the first holder of which is Sir Clement Price Thomas. Some of the duties of the Adviser were originated by Sir Gordon Gordon-Taylor and are being continued by Sir Clement; these include advice to young surgeons regarding their future surgical training.

CHANGE OF ADDRESS

Subscribers should notify the Canadian Journal of Surgery of their change of address *two* months before the date on which it becomes effective, in order that they may receive the Journal without interruption. Coupon on page 25 is for your convenience.

FORTHCOMING MEETINGS

47TH ANNUAL CLINICAL CONGRESS: THE AMERICAN COLLEGE OF SURGEONS

More than 11,000 surgeons and physicians from the United States, Canada and many other countries are expected to attend the 47th Annual Clinical Congress of the American College of Surgeons in Chicago, October 2 to 6, 1961. Approximately 1000 doctors will participate in the program which will include nine postgraduate courses, 258 new research reports from medical centres throughout the country, 68 medical motion pictures, 26 cine clinics, 14 operation telecasts from Billings Hospital of the University of Chicago, and 300 scientific and industrial exhibits. Headquarters of the Congress will be located in the Conrad Hilton Hotel.

Major addresses will be delivered by Dr. Robert M. Zollinger of Ohio State University College of Medicine, the incoming president of the College, who will speak on the subject of "Surgical Tithing"; Dr. Francis D. Moore of Harvard Medical School, who will deliver the annual Baxter Lecture on "The Control of Effective Volume and Tonicity: Body Composition", and Dr. Preston A. Wade of Cornell University Medical College, who will present the annual Trauma Oration, "The Specialist and the Injured Patient". This year's historic Martin Memorial Lecture will be delivered by Admiral Hyman G. Rickover.

Nine postgraduate courses will be devoted to the following topics: Pre- and Postoperative Care; Gastrointestinal Disease; Diseases of the Liver, Biliary Tract and Pancreas; Cardiovascular Surgery; Injury of Joints of the Upper Extremity; Obstetrics and Gynecology; Cancer Chemotherapy; Thoracic Surgery; and Recent Advances in Pediatric Surgery.

Additional sessions will provide for discussions on What's New in Surgery; Neurological Surgery; Gastrointestinal and Biliary Diseases; Plastic Surgery; Tumours; Anesthesia and Pulmonary Problems; Cardiovascular Surgery, and Orthopedic Surgery.

Panel discussions will feature symposia on trauma and cancer, and individual reports will be presented by a broad range of experts on the subjects of thyroid disease, emergency department problems in trauma, facial injuries, skin grafts, viral infections in pregnancy, knee joint fractures, duodenal ulcer, diseases of the spleen, cerebrovascular insufficiency and renal tumours.

For further information write direct to the American College of Surgeons, 40 East Erie Street, Chicago 11, Ill.

THE SOUTH WESTERN ONTARIO SURGICAL ASSOCIATION

The Annual Meeting of the South Western Ontario Surgical Association will be held in the Busby Memorial Lecture Room at the Victoria Hospital, London, Ont., on Wednesday, October 25, 1961. The meeting will begin at 9 a.m. and Professor Charles Rob of Rochester, N.Y., will be the guest speaker.

For further information write to Dr. Robert M. McFarlane, Secretary, Surgery Office, Victoria Hospital, London, Ont.

THE CANADIAN SOCIETY FOR THE STUDY OF FERTILITY

The eighth Annual Meeting of the Canadian Society for the Study of Fertility will be held at the Sheraton-Brock Hotel, Niagara Falls, Ont., on October 27 and 28, 1961.

For further information write to the Secretary, Dr. George H. Arronet, Infertility Centre, Royal Victoria Hospital, Montreal.

CANADIAN HEART ASSOCIATION AND NATIONAL HEART FOUNDATION OF CANADA

The joint Annual and Scientific Meetings of the Canadian Heart Association and the National Heart Foundation of Canada will be held in Vancouver from November 13 to 18, 1961.

For further information write to Dr. John B. Armstrong, National Heart Foundation of Canada, 501 Yonge Street, Toronto 5.

EIGHTH INTERNATIONAL CANCER CONGRESS

The Eighth International Cancer Congress will take place at the Moscow State University, Moscow, from July 22 to 28, 1962, under the auspices of the International Union Against Cancer.

Problems concerning cancer will be approached at the Congress from both experimental and clinical aspects.

Applications for the reading of papers will be considered only on condition that both the application and abstract of the paper (not exceeding 250 words) are submitted before November 1, 1961.

The registration fee is $30.00 (U.S.) per member, if sent before April 1, 1962.

Foreign members of the Congress will be serviced by the Soviet travel agency "Intourist".

Information concerning the Congress, registration forms, and applications to read papers and to show films, may be obtained from the General Secretary of the Soviet National Organizing Committee, Professor L. Shabad, or the Assistant General Secretary, Dr. N. Perevodchickova, Academy of Medical Sciences of the U.S.S.R., 14 Solyanka, Moscow, U.S.S.R.

FOURTH WORLD CONGRESS OF CARDIOLOGY

The Fourth World Congress of Cardiology will be held at the Medical Center, Mexico City, from October 7 to 13, 1962. The President of the Committee is Dr. Ignacio Chavez.

Further information may be obtained from the General Secretary, Dr. Isaac Costero, IV World Congress of Cardiology, Institute N. de Cardiologia, Avenida Cuauhtemoc 300, Mexico 7, D.F.

THIRD INTERNATIONAL CONGRESS OF PLASTIC SURGERY

The Third International Congress of Plastic Surgery will be held at the Statler Hilton Hotel, Washington, D.C., from October 13 to 18, 1963.

Inquiries should be addressed to the Secretary-General, Leslie H. Backus, M.D., 217 Linwood Avenue, Buffalo 9, N.Y., U.S.A.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Abdominal Surgery. Edited by Arthur W. Allen and David Woolfolk Barrow. 658 pp. Illust. Paul B. Hoeber, Inc., New York, 1961. $21.50.

The Closed Treatment of Common Fractures. 3rd ed. John Charnley. 272 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $8.50.

La Grosse Tubérosité de l'Estomac. T. Schops. 575 pp. Illust. Gaston Doin & Cie, Paris, 1961. 125 NF. $25.00 (approx.).

Orthopaedic Approaches: A Stereographic Manual. John J. Joyce, III, and Michael Harty. 80 pp. Illust. The Williams & Wilkins Company, Baltimore, Md., 1961. $28.00.

(Continued on page 501)

BOOK REVIEWS

(See also pages 464, 472, 482 and 489)

THE SURGICAL TREATMENT OF PORTAL HYPERTENSION, BLEEDING ESOPHAGEAL VARICES AND ASCITES. M. Judson Mackby, Kaiser Foundation Hospital, San Francisco. 277 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $11.50.

The author has presented a commendable review of the subjects embraced in his title. Diagnosis, clinical assessment, and both emergency and elective treatment of these problems are thoroughly considered. The technique and indications for esophageal tamponade, variceal ligation, and the various shunting procedures are fully discussed.

In addition to presenting his own clinical experience in this field, the author has made a nation-wide survey of many of the recognized authorities on portal hypertension in an attempt to elucidate some of the more controversial problems.

Splenic manometry is only referred to briefly and its potential is not suggested. It would seem that a more precise discussion of the physiopathology and hemodynamics of portal hypertension would add to the value of a monograph of this type.

While the author is obviously not impressed with the results of shunt surgery for ascites, the current interest in this phase of portal hypertension might warrant a more critical evaluation of its possibilities.

The book is well written, well illustrated and includes an extensive bibliography.

MODERN TRENDS IN UROLOGY (SECOND SERIES). Edited by Sir Eric Riches, Past President, British Association of Urological Surgeons (Home and Overseas); Surgeon and Urologist, Middlesex Hospital, London. 287 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1960. $14.00.

This book provides an excellent comprehensive survey of the major urological advances of recent years. Twenty-seven authorities have contributed to a text of under 300 pages. Subjects which are not found in standard urological texts are included. The principles and use of the image intensifier are described. Surgery of the renal artery is reviewed. Two chapters are devoted to renal transplantation, a subject of major interest to all urologists. Four authors have described the use of intestinal segments in urology. Included is a reappraisal of the existing classifications of testicular tumours.

This text is particularly recommended to students of all specialties preparing for the higher examinations.

SURGICAL DISEASES OF THE PANCREAS. John M. Howard, M.D., F.A.C.S., Department of Surgery, Hahnemann Medical College; Surgeon-in-Chief, Hahnemann Hospital; Consultant in Surgery, Walter Reed Army Medical Center, and George L. Jordan, Jr., M.D., M.S., F.A.C.S., Associate Professor of Surgery, Baylor University College of Medicine. 607 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1960. $20.00.

This is an authoritative and timely volume, the result of a critical survey of the world literature and of the experience of its authors. One half the book is the product of the senior author whose main interest has been the various aspects of pancreatitis, while Dr. Jordan has contributed the chapters on cysts and tumours. Some nine other authors have provided chapters on the contributory subjects of anatomy, physiology and laboratory diagnosis which are treated in orthodox fashion, with stress on the current interest in the structure of the distal ends of the hepatic and pancreatic ductal systems. Implicated as it is with physiology, the discussion on the laboratory diagnosis of pancreatic disease is of particular interest. The major single aid to diagnosis remains the test for serum amylase. Its limits and the importance of its interpretation in the light of the clinical findings is stressed. The facts and fancies about the cause of pancreatitis are discussed; the theory of the reflex flow of bile is not accepted and the mechanism remains suppositious. Meconium ileus is dealt with at disproportionate length, but this may be excused by the excellence of the article. Pancreatitis is a major interest. A convincing distinction is drawn between pancreatitis of gallstone origin and alcoholic pancreatitis, a distinction which should be the basis of classification except for those much fewer traumatic, metabolic or postoperative cases. Biliary disease is responsible for one-half the cases and in these, subsequent treatments of the biliary tract disease is adequate for the prevention of recurrences, whereas in alcoholic pancreatitis such operative treatment has no place, recurrences are to be expected and pathological changes in the organ leading to diabetes or even calcification are not uncommon. This is argued from adequate evidence. Of interest is the occasional occurrence of massive intestinal hemorrhage in pancreatic disease, particularly in the Zollinger-Ellison syndrome. It is hinted that in the presence of such otherwise unexplained hemorrhage, disease of the pancreas should be sought for. In the treatment of pseudocysts of the pancreas a distinct preference for internal drainage is expressed, but this should depend on the ability of the surgeon concerned. The knowledge of islet-cell tumours has been made current by a tabular presentation of 1000 cases and this and the 2000 references divided amongst the various chapters, add greatly to the bulk of the volume. The discussion of pancreatic tumours is of necessity factual, but complete. A chapter on partial and total pancreatectomy discusses the various modifications of these still formidable procedures and validates conclusions by statistical evidence. One is impressed by the scientific method in the collection and analysis of such an immense amount of material. The book should be readily available as pancreatic disease is not sufficiently frequent for all surgeons to be fully conversant with its problems.

PRINCIPLES OF GENERAL NEUROPHYSIOLOGY RELATING TO ANAESTHESIA AND SURGERY. Barry D. Wyke, Senior Lecturer in Applied Physiology, Royal College of Surgeons of England. 136 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1960. $4.25.

The author states in the preface that this book is a reprint of Chapter 5 of volume I of "General Anaesthesia", edited by Evans and Gray, and is presented in this form following many requests that the information should be made available to a wider group of readers than anesthetists, for whom it was originally intended.

A concise, but inclusive review of cellular neurophysiology including the known effects of anesthetic agents on neurons forms the first section. This is followed by an excellent discussion of interneuronal communication and the interpretation of the recorded electrical activity of the brain, particularly as it is affected by varying depths of general anesthesia. The concluding section deals with the cerebral circulation, metabolism and the cerebral spinal fluid, and how anesthetics affect these. Several well chosen microphotographs and a number of diagrams add much to the clarity of the description.

Although no index is provided, the table of contents is much more detailed than is usually found in a volume of this size, and names all the special topics that are discussed or described. The three main sections of the book conclude with an up-to-date selected bibliography of the key works on each subject.

The purpose of this book is to explain the controlled reversible intoxication of the nervous system by anesthetic agents, so that the anesthetist may be able to regulate these changes better through his more detailed understanding and evaluation of observed alterations. Such information helps the anesthetist to develop his art as well as to strive towards making his work a more precise science. The author has succeeded in presenting a difficult subject in an interesting way that is easy to follow. This area of knowledge is one in which anesthetist and surgeon still have much to learn; they would profit greatly by reading this book.

MODERN TRENDS IN CARDIAC SURGERY. Edited by H. R. S. Harley, Consultant Thoracic Surgeon, United Cardiff Hospitals and Welsh Regional Hospital Board. 292 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1960. $14.00.

It is a courageous undertaking to edit a book on cardiac surgery at this particular time, when the picture is changing so rapidly. A matter of one year may see not only the introduction of new techniques but the application of new principles in cardiac surgery.

British writers have always been lucid and clear and this book is no exception. It is a neat, well-organized and easily read text without too much detail. It is written in the manner of a textbook; each section is written by a British authority, with a good deal of space devoted to surgical anatomy, clinical features and diagnosis. It would be more easily read if some authors had made greater use of illustrations. This is particularly true of the descriptions of operative techniques where the amount of detail varies from section to section.

The book is small and thus represents a very easy reference. Its compactness is obviously the result of careful and thoughtful selection of topics of practical value. One might suggest that a section dealing with all the new techniques of investigation and another discussing the important function of intensive postoperative care units would provide completeness and clarity.

It is inevitable that during the time required to edit and publish such a book, some of the most recent developments in the field are missed, such as new open-heart methods of treating acquired valvular disease and local deep hypothermia of the heart. However, until these techniques have been tested and tried this is not a serious omission in a reference text of this kind.

The editor has wisely not attempted to encompass too broad a scope. The book is well proportioned and, as the introduction indicates, properly directed to the general medical consultant, the trainee and the general practitioner. In spite of this there are some excellent sections, particularly the ones on extracorporeal circulation by Melrose and deep hypothermia by Drew, which are of interest to everyone, including the cardiac surgeon.

KLINISCHE CHIRURGIE FUER DIE PRAXIS. In vier Bänden. Band I, Lieferung 4 (Clinical Practice of Surgery. In 4 volumes Vol. I, Part 4). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 366 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1960. $12.85.

This section begins with a monograph on the subject of allergy. It discusses the theories of allergy, anaphylaxis, immunity, diagnostic procedures and methods of treatment. Although the long list of different allergic conditions does include a few "surgical" diseases, the inclusion of this chapter in a surgical book is a rare innovation. Three short chapters present a concise account of the surgery related to childhood, pregnancy and geriatrics. The important part of this section is an essay on the central, autonomic, spinal, and vegetative nervous systems that is easy to read and is up-to-date.

BRITISH MEDICAL BULLETIN. Hypothermia and the effects of cold. Vol. 17. No. 1. Scientific Editor, Dr. A. S. Parkes, F.R.S. Introduction by Dr. O. G. Edholm. Symposium of 14 contributions presenting the experience and opinions of 16 British workers actively engaged in expanding the frontiers of knowledge in this field. 75 pp. Illust. Medical Department, The British Council, 65 Davies Street, London, W.1., 1960. $3.25.

This symposium published in the British Medical Bulletin entitled "Hypothermia and the Effects of Cold" has given broad coverage to this very interesting subject. Probably the most interesting parts are those related to fundamental studies in animals by Dr. Salt concerning resistance of poikilotherms, and the studies in hibernation by Harrison Matthews. This review is far too technical for the average doctor not specifically interested in the problem of hypothermia. However, there is a wealth of material on the various aspects of the surgical use of hypothermia, especially as demonstrated by Mr. Drew in his study of the use of profound hypothermia in cardiac surgery. There is some question whether his form of hypothermia is ideal; and conflicting reports in the literature as to the injurious effects of profound hypothermia on the brain, in other surgeons' experience, may mitigate against his postulates. Unfortunately he does not break down his exact mortality in the use of profound hypothermia, but he does indicate that it compared favourably with the other methods. Dr. Bigelow's review of the problem of hormones in hypothermia was stimulating indeed and highly complex. Dr. Lougheed gave a review of the present-day knowledge of the nervous system in hypothermia which seemed to be complete and informative.

All in all it was rather disappointing to see such a mixture of the accounts of the surgical use of hypothermia and the basic biochemical hormonal changes in the poikilotherm and the hibernating animal. The changes are so different in the poikilotherm subjected to hypothermia when compared with the hibernating state, that in a single symposium it represents a rather wide diversification of knowledge. However, to the expert in the broad field of hypothermia it undoubtedly served a purpose, but to the practising surgeon or medical man it was much too complex a report.

(Continued on page 498)

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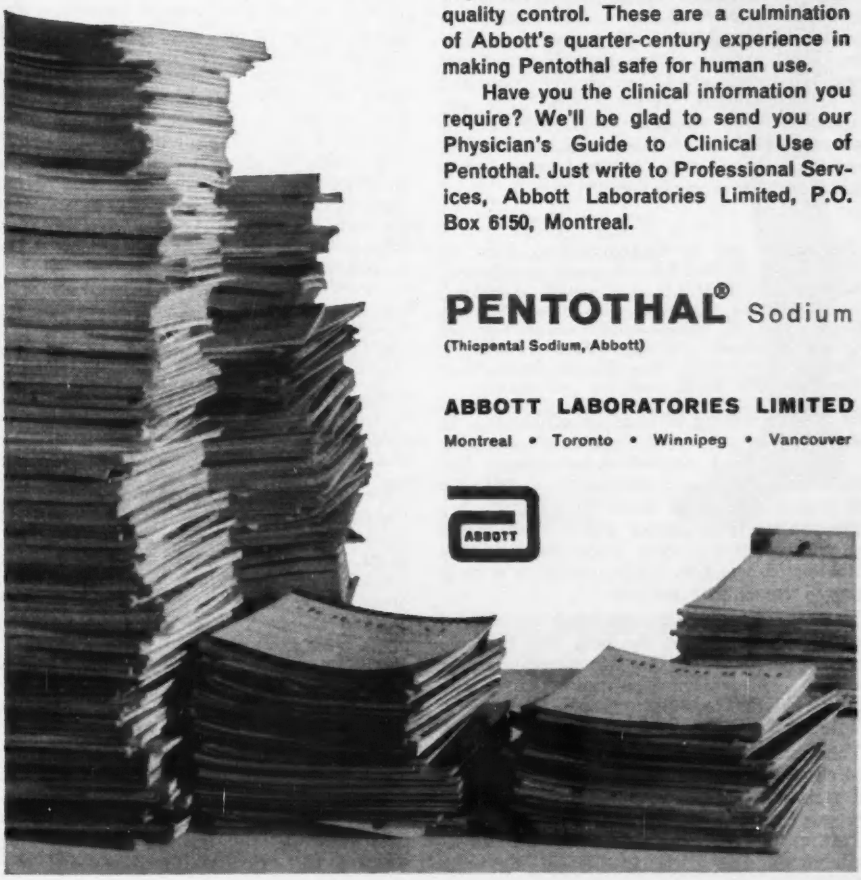
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(Continued from page 493)

THEORY OF SHOULDER MECHANISM. A. K. Saha, Professor of Surgery, Nilratan Sircar Medical College. Foreword by Edward L. Compere, M.D., Professor and Chairman of Orthopedic Surgery, Northwestern University Medical School, Chicago. 107 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $6.00.

This book is another publication from Charles C Thomas in a valuable line of highly specialized monographs. Though these monographs obviously do not aspire to become monumental textbooks, they do represent the sincere efforts of dedicated medical scientists to present their intense personal views and research before the medical public. Therefore, for making these monographs available, Mr. Thomas (who is a real person and not a corporation) must be congratulated as much as the authors. Such publishing ventures surely constitute a risky business financially and are better described as public service.

Dr. Saha's little book presents his views on shoulder mechanisms lucidly and succinctly. The style is spare and direct. The "science" is usually easy to follow and generally acceptable. The surgical views, though sometimes unorthodox and novel, are clearly presented and ably defended. Surgeons who have any dealings with the shoulder joint must become conversant sooner or later with Dr. Saha's views and there is no pleasanter way than reading his interesting book.

RADIOLOGY AS A DIAGNOSTIC AID IN CLINICAL SURGERY. Howard Middlemiss, M.D., F.F.R., D.M.R.D., Director of Radiology, United Bristol Hospitals, Lecturer-in-Charge, Department of Radiodiagnosis, University of Bristol, England. 150 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $9.00.

This is a refreshing little book which departs from the usual pattern of a digest of radiology for surgeons and general practitioners. The author is a practising diagnostic radiologist who makes a plea for more co-operation between the referring doctor and the radiologist and points out in many instances how the radiologist can modify his examination if he is aware of the clinical problem.

The broad field of radiology is covered adequately, illustrating the help that the surgeon can expect from the various examinations available. The author also points out many conditions which are not possible to diagnose radiologically.

The reviewer believes this book should be read carefully by all surgeons who teach medical students. It will also be helpful to all surgeons and general practitioners practising in centres where good radiological service is available.

ACTUALITES SUR LES MALADIES DES VEINES. Varices, Phlébites, Hémorroïdes. Colloque tenu dans le service du Professeur André Sicard, l'Hôpital Beaujon, Paris, April 1958. 89 pp. Illust. L'Expansion Scientifique Française, Paris, 1960. [illegible]. $2.60 (approx.).

Ce colloque de phlébologues, tenu dans le service du Professeur André Sicard, est bien présenté. C'est une rapide et concise revue des idées actuelles sur les maladies des veines, tant au point de vue diagnostic que thérapeutique. Les divers auteurs exposent leurs vues et les résultats qu'ils ont obtenus. La thérapeutique est, de façon générale, divisée en médicale et chirurgicale.

Les diverses techniques sont discutées, l'emphase étant sur le traitement médical. On n'est toutefois pas assez précis sur les résultats, et on insiste trop peu sur les récidives et surtout le danger des complications, en particulier la phlébite profonde d'origine chimique (produit injecté). Du côté chirurgical, on ne parle pas des accidents per-opératoires et de leurs désastreuses conséquences pouvant aller jusqu'à l'amputation. Apparemment on sclérose les varices sur une plus grande échelle en France qu'en Amérique du Nord, puisque "un malade sur dix seulement", relève de la chirurgie.

En conclusion, Tubiana dit que "le traitement des varices est loin d'avoir la simplicité que certains lui prêtent et ne saurait rester confiné à une seule méthode". Fort vrai, et c'est dans le choix judicieux du traitement qu'originent les bons résultats. Les clichés et photographies sont excellents et la présentation générale du volume est bien. Il y a beaucoup d'idées et d'informations à retenir et l'ouvrage vaut la peine d'être lu.

KLINISCHE CHIRURGIE FUER DIE PRAXIS. In vier Bänden. Band III, Lieferung 3 (Clinical Practice of Surgery. In 4 volumes. Vol. III, Part 3). Edited by O. Diebold, H. Junghanns and L. Zukschwerdt. 221 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $9.75.

There are three excellent monographs in this instalment of the *Clinical Practice of Surgery* series. The first presents a discussion of diseases, injuries, and malformations of the small bowel, with an additional chapter on bowel obstruction. The second covers a similar field with reference to the colon, and the third deals with the subject of appendicitis.

The description of different methods of clinical examination, and the adequate and lucid discussions of anatomy and pathophysiology follow the pattern set in the other volumes of this series. Like the previous instalments, this book is up-to-date and contains ample references to the current literature.

This section is a very useful and valuable component of the *Clinical Practice of Surgery* series.

SYNOVIAL JOINTS: THEIR STRUCTURE AND MECHANICS. C. H. Barnett, D. V. Davies and M. A. MacConaill. 304 pp. Illust. Longmans, Green & Co. Ltd., London; J. B. Lippincott Company, Montreal, 1961. $8.50.

Coming as it does after a recent flood of publications on special subjects that (in spite of their book-jacket "blurb") can only interest very select groups of readers, this wonderful book is particularly welcome. Though it confines itself to the consideration of only one system, it does so with such intelligence, vivacity and charm that it will almost certainly captivate any physician or surgeon who has even the slightest interest in synovial joints. Oddly enough, it also brings forth a considerable amount of new information for specialists in basic sciences and research workers, who therefore cannot ignore it.

Such books—capable of holding the interest of both specialists and non-specialists—have become all too rare in medical publishing, particularly in the field of arthrology and kinesiology. In retrospect, one may guess that perhaps the omens in the case of "Synovial Joints" were all favourable. For example, the authors are English, Welsh and Irish and therefore their language is only slightly blighted with American "scientific" jargon. Moreover, all three have been actively engaged in the study of joints for many years, and they are actively engaged in the teaching of undergraduate medical students in London and in Cork.

"Synovial Joints" has a wide scope. The articular cartilage, synovial membrane, ligaments and other components are described in relation to their functions, with reference to the chemical composition of the tissues and with the clinical application of new information constantly stressed. There is a full discussion of the role of synovial joints in body movement and posture (a field in which both Barnett and MacConaill are acknowledged authorities), nutrition and metabolism of synovial joints, their development (Davies' special field), age changes, physical properties and degenerative changes. The writing is so well blended that it is impossible to determine which author is responsible for the various chapters. Nonetheless, the special contributions of each author to arthrology receive excellent and unexaggerated representation.

The book is well illustrated with good photomicrographs, drawings and diagrams. The selective bibliography is particularly noteworthy and to many doctors it should be an adequate incentive for becoming acquainted with "Synovial Joints".

In conclusion, this reviewer feels honoured that (after years of routine labour) he has again had the rare opportunity of reviewing what he considers will become a modern "classic" in medical literature.

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(Continued from page 494)

Cardiac Surgery 1960-61. Edited by Charles P. Bailey, Chairman and Professor of Surgery, New York Medical College. 179 pp. Illust. Davis Monograph Series. F. A. Davis Company, Philadelphia; The Ryerson Press, Toronto, 1960. $5.50.

Chirurgie des Voies Biliaires Extra et Intra-Hépatiques (Surgery of the Extra- and Intrahepatic Bile Ducts). Claude Olivier, Professeur agrégé, Faculté de Médecine de Paris, Chirurgien de l'Hôpital Tenon. 612 pp. Illust. Masson & Cie, Paris, 1961. 100 NF. $20.00 (approx.).

Die Chirurgische Behandlung der Angeborenen Fehlbildungen (The Surgical Management of Congenital Abnormalities). Edited by K. Kremer, Düsseldorf. 592 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $29.50.

Geochronology of Rock Systems. Annals of the New York Academy of Sciences. Vol. 91, Art. 2, pages 159-594. Edited by Franklin N. Furness. Illust. New York Academy of Sciences, New York, April 1961.

Le Kyste Hydatique (The Hydatid Cyst). Thérapeutique chirurgicale. P. Goinard, Professeur de Clinique thérapeutique chirurgicale, Faculté de Médecine d'Alger; J. Pegullo, Chirurgien des Hôpitaux d'Alger, et G. Pélissier, Chirurgien-assistant des Hôpitaux d'Alger. 204 pp. Illust. Masson & Cie, Paris, 1960. 45 NF. $9.00 (approx.).

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery. Vol II. Rodolfo Cosentino, M.D., Assistant Professor in Orthopedic Surgery, University of La Plata, Argentina. Preface by Carroll B. Larson, M.D., Professor and Head of Department of Orthopedic Surgery, State University of Iowa. 264 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1960. $15.50.

An Atlas of Pain Patterns. Sites and behavior of pain in certain common diseases of the upper abdomen. L. A. Smith, N. A. Christensen, N. O. Hanson, D. E. Ralston, R. W. P. Achor, K. G. Berge, G. W. Morrow, Jr., and A. H. Bulbulian, Mayo Clinic and Mayo Foundation, Rochester, Minnesota. 54 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $13.75.

The Surgery of Mitral Stenosis. Robert P. Glover and Julio C. Davila, University of Pennsylvania School of Medicine, Philadelphia. 219 pp. Illust. Grune & Stratton, Inc., New York; The Ryerson Press, Toronto, 1961. $10.50.

Roentgenology of Intracranial Meningiomas. Sidney P. Traub, Associate Professor of Radiology, University of Saskatchewan College of Medicine. 238 pp. Illust. Charles C Thomas, Springfield, Ill., 1961. $14.00.

The Spinal Cord. Basic Aspects and Surgical Considerations. George Austin. 532 pp. Illust. Charles C Thomas, Springfield, Ill., 1961. $26.50.

(Continued on page 502)

OPERATIVE SURGERY

General Editors: CHARLES ROB, M.C., M.Chir, F.R.C.S. Professor of Surgery, St. Mary's Hospital, London, and RODNEY SMITH, M.S., F.R.C.S. Surgeon, St. George's Hospital, London.

This work which was originally published as a complete set of eight volumes and index has now been specially edited and divided into groups shown below. Each volume contains its own index and in the case of sets of volumes, there is a comprehensive index to the whole set.

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(Continued from page 501)

Surgery of the Anus, Rectum and Colon. J. C. Goligher. 829 pp. Illust. Cassell & Co. Ltd., London; British Book Service (Canada) Ltd., Toronto, 1961. $30.25.

Les Syndromes Abdominaux Aigus Pseudo-Chiurgicaux. Henri Merle. 272 pp. 8 plates. Gaston Doin & Cie, Paris, 1961. 28 NF. $5.60 (approx.).

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VOL. 4, NO. 5—OCTOBER 1961

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ORIGINAL ARTICLES

ETUDE CLINIQUE DE 238 CAS D'ENDOMETRIOSE CHIRURGICALE*

BERNARD LAMBERT, M.D.,† PIERRE MEUNIER, F.R.C.S. et CHARLES OUIMET, M.D., *Montréal*

CET ARTICLE étudie 238 cas d'endométriose chirurgicale traités à l'Hôtel-Dieu de Montréal pendant une période de 10 ans, de janvier 1950 à janvier 1960, et à propos desquels les constatations et le diagnostic ont été faits, cliniquement ou le plus souvent, par biopsie-exérèse, ou par l'étude des pièces elles-mêmes. Dans la grande majorité des cas, le laboratoire de pathologie avait confirmé le diagnostic antérieur et nous avons éliminé les kystes hémorragiques du corps jaune, facilement confondus avec les kystes endométriotiques. Les cas d'adénomyose utérine ont été compilés intentionnellement avec l'endométriose pelvienne en considérant deux faits: le premier, qu'au moins 30 cas sur les 238 (12%) étaient porteurs d'endométriose combinée externe et interne; ceci confirme les chiffres de Novak et de Norwood;[1] le second étant que la localisation la plus fréquente d'endométriose, après celle de l'ovaire, était l'utérus bien avant le péritoine pelvien ou les ligaments utéro-sacrés. Nous avons observé 200 cas d'endométriose ovarienne, 54 cas d'endométriose utérine, 37 cas de Douglas ou des ligaments utéro-sacrés. Les cas d'endométriose interne étaient classés comme tels, par nos pathologistes lorsque le 1/3 externe de la paroi utérine était seul atteint. Nous n'avons pas inclus les cas d'endométriose stromale étant donné leur extrême rareté; nous verrons quelles sont les variations de pourcentage et d'interprétation, dues à l'adjonction de l'adénomyose. Le service de gynécologie a opéré les 2/3 des malades et le reste a été opéré par le service de chirurgie générale. Nous exposerons d'abord les sept facteurs les plus fréquemment invoqués dans la pathogénie de l'endométriose; nous verrons ensuite ce que l'on peut retenir en conclusion.

PREMIER FACTEUR.—*Facteur social et civil*

L'étude du statut social des patientes a été beaucoup plus révélatrice que celle du statut civil et s'est superposée aux données classiques. Le pourcentage des cas privés était nettement supérieur aux cas semi-privés et aux cas publics. En effet, 32% étaient privés, 60% semi-privés et seulement 8% étaient en salle, soit 18 cas d'assistance publique formant une moyenne de 1.8 cas par année. Si nous considérons le fait que nous possédons un lit privé pour deux lits publics, la prépondérance des patientes privées est évidente. L'un des facteurs déterminant de cette différence pourrait être la forte variation du taux de fertilité selon le statut social, la classe privilégiée faisant preuve d'un taux de fertilité moindre. De nombreux auteurs admettent la relation infertilité-endométriose, mais soulignons ici l'opposition radicale des points de vue. Certains prétendent que la stérilité volontaire est cause d'endométriose par la succession ininterrompue de cycles mentruels amenant une irritation des restes d'épithelium cœlomique ou un reflux sporadique de sang dans la cavité péritonéale; d'autres prétendent que l'endométriose est cause de stérilité involontaire, par la dyspareunie haute, la rétroversion, les modifications possibles du péristaltisme tubaire dans l'endosalpingose, le recouvrement des pavillons par les fausses membranes ou leur cloisonnement, l'atteinte ou la destruction du parenchyme ovarien. Nous avons trouvé qu'en général, le taux de fertilité, s'il est de 83 à 86% dans les classes moyennes, s'élève jusqu'à 91 à 94% dans les classes populaires et rurales.[2]

Le statut civil de nos patients fut beaucoup moins révélateur, la proportion de

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*Communication donnée au 30ème Congrès du Collège Royal des Médecins et Chirurgiens du Canada, Ottawa, le 21 janvier 1961.

Service de Gynécologie de l'Hôtel-Dieu de Montréal.

†Adresse actuelle: Gynecological-Pathological Laboratory, Johns Hopkins Hospital, Baltimore, Md.

femmes mariées et de célibataires étant de 74 à 26%, taux comparable à ceux de Meigs[3] de 74.1% de femmes mariées parmi les cas d'endométriose et de 74.3% de femmes mariées dans une population normale et saine. Le facteur célibat est donc moins déterminant, que le mariage stérile chez les classes privilégiées. Cette hypothèse extrêmement intéressante mériterait certainement une investigation plus poussée, mais assez difficile du fait que le questionnaire sur la stérilité et éventuellement sur le type de moyen anticonceptionnel employé, demeure impossible. Les patientes donnent rarement des réponses conformes à la réalité.

Deuxième Facteur.—*Facteur âge*

L'âge moyen d'apparition de l'endométriose est de 30 à 34 ans, mais nous pouvons noter deux faits. Le premier, c'est qu'après 50 ans l'incidence d'endométriose tombe brusquement à quatre cas et qu'elle suppose une ménopause tardive (Tableau I).

TABLEAU I.—Age

Age	*Total*
15 - 19	5
20 - 24	20
25 - 29	37
30 - 34	59
35 - 39	49
40 - 44	41
45 - 49	23
50 et plus	4
Total	238

Robichaux, Sanders et Nicholson[4] citent un cas rarissime actif paru sept ans après la cessation des règles: une femme de 50 ans opérée pour hémopéritoine secondaire à la rupture d'un énorme kyste endométriotique; cette patiente était ménopausée depuis l'âge de 43 ans. Rien ne prouve cependant que ce kyste ne datait pas de la vie génitale de la malade. Des quatres quinquagénaires, deux d'entre elles étaient porteuses d'endométriose externe, une d'endométriose combinée, et une d'endométriose interne isolée. Aucune n'était ménopausée. Deuxième fait, c'est qu'un total de 25 patientes avaient 24 ans ou moins (dont cinq au dessous de 19 ans) contrairement à ce qu'a rapporté Meigs.[5] Norwood[1] sur 220 cas, donne des âges variant de 15 à 62 ans. Les 25 patientes avaient toutes de l'endométriose ovarienne et sept d'entre elles une endométriose du Douglas, des utéro-sacrés, et une du vagin. Aucune n'avait un utérus augmenté de volume ce qui diminue les chances d'endométriose interne, quoiqu'on n'en ait eu aucune preuve microscopique, vu l'absence de biopsie du muscle utérin.

TABLEAU II.—Age-parité

Age	*Nullipares*	*Multipares*
15 - 19	5	0
20 - 24	15	3
25 - 29	23	8
30 - 34	29	19
35 - 39	22	19
40 - 44	18	16
45 - 49	5	15
50 et plus	1	3
Total	118	83

Troisième Facteur.—*Age-parité*

Si nous examinons maintenant le facteur âge en rapport avec la parité, il y a une proportion de deux multipares pour trois nullipares, l'âge optimum pour les deux catégories demeure le même avec une légère prédominance des multipares pour un degré d'âge plus avancé (Tableau II).

Toutes les patientes de moins de 19 ans étaient nullipares, mais on trouve trois des 18 patientes entre 20 et 24 ans qui sont multipares. La différence des deux patientes d'avec la somme totale (Tableau I) est expliquée par les cas de patientes mariées dont l'état obstétrical n'avait pas été relevé. La prépondérance des multipares est manifeste après 45 ans, à cause de l'incidence plus grande d'adénomyose ou d'endométriose mixte avec l'élévation de l'âge et celle-ci étant plus élevée avec les grossesses (Tableau III).

TABLEAU III.—Adénomyose-endométriose

Age	*Adénomyose*	*Rapport*	*Endométriose*
25 - 29	2	1/18	37
30 - 34	13	2/9	59
35 - 39	13	2/7	49
40 - 44	12	1/3	41
45 - 49	12	1/2	23
50 et plus	2	1/2	4

Quatrième Facteur.—*Facteur fertilité*

Ceci nous amène à étudier le facteur

fertilité des femmes mariées qui est de 57%. Nous avons laissé de côté les célibataires, la plupart du temps sans enfants ce qui porte le pourcentage absolu à environ 40%. Il y a nette diminution des cas après cinq enfants, la quatrième grossesse constituant la plaque tournante du système. Meigs[6] rapporte des incidences de fertilité de femmes mariées de 63 à 68%, alors qu'on cite dans la littérature des chiffres de 44 à 79% de fertilité[1] (Tableau IV).

TABLEAU IV.—FERTILITÉ (57%)

73 patientes dont	24	1	enfant
	19	2	enfants
	19	3	"
	11	4	"
8 patientes dont	3	5	"
	4	6	"
	1	7	"
3 patientes dont	1	10	"
	1	13	"
	1	16	"

Nous pourrons distinguer trois sous-groupes. Le premier est de 73 malades ayant de un à quatre enfants, ce qui nous laisse entendre une certaine fréquence de la maladie dans les petites familles; un second groupe de huit patientes ayant de cinq à sept enfants, et un troisième groupe de trois patientes ayant plus de 10 enfants. Les mères de 13 à 16 enfants avaient toutes deux au-delà de 45 ans et présentaient uniquement de l'adénomyose utérine. Notre pierre d'achoppement fut la multipare de 10 enfants, mariée à 16 ans, porteuse d'un kyste endométriotique de l'ovaire gauche, asymptomatique, qui aurait consulté pour des ménorragies dues à une sub-involution chronique de l'utérus et endométrite, ce qui nous laisse absolument ignorants du moment d'apparition du kyste. Il est possible que la patiente en ait été porteuse depuis assez longtemps, vu l'absence de symptômes spécifiques. Quatre des huit multipares de cinq à sept enfants présentaient de l'endométriose externe. En somme, nous constatons que la fertilité d'endométriose est sûrement inférieure à celle d'une population normale, mais qu'il reste tout de même un groupe assez important de patientes ayant moins de quatre enfants.

CINQUIÈME FACTEUR.—*Type d'endométriose*

Si nous étudions maintenant comparativement les types d'endométriose, nous constatons que l'adénomyose seule chez les nullipares est une rareté, qu'il y a sûrement diminution proportionnelle de l'endométriose externe avec les accouchements, avec l'apparition de l'adénomyose; ce phénomène était considéré par Hertig comme presque normal après plusieurs grossesses. Nous avons pris grand soin cependant, de ne pas inclure dans notre étude les endométrites, les sub-involutions chroniques de l'utérus et les scléroses élastigènes (Tableau V).

TABLEAU V.—TYPES D'ENDOMÉTRIOSE-PARITÉ

Type	*Nullipares*	*Multipares*
Endométriose externe, mixte, Douglas, utéro-sacrés, etc.	114	64
Endométriose interne	4	16
Divers	—	3
Total	118	83

SIXIÈME FACTEUR.—*Temps depuis dernier accouchement—Age au mariage*

Meigs a énormément insisté sur le temps écoulé depuis le dernier accouchement comme étant propice à l'éclosion d'une endométriose externe de même que les mariages tardifs. Sur une série de 105 malades sur 112, il rapportait des stérilités secondaires de 17 ans ou plus. Sur les 37 malades dont nous avons retracé la date du dernier accouchement nous avons distingué trois groupes:

1. Un groupe de 10 malades ayant accouché depuis trois ans ou moins, porteuses uniquement d'endométriose externe ou mixte, ce qui nous permet de nous poser des questions, quant à l'influence directe retardante de la grossesse sur une endométriose évolutive;

2. Un second groupe de 13 malades ayant accouché depuis quatre à six ans, dont deux présentaient uniquement de l'adénomyose;

3. Un dernier groupe de sept ans ou plus, de 14 malades et comportant le même nombre d'adénomyoses que le second groupe. L'âge lors du mariage n'a pas été

TABLEAU VI.—Age lors du mariage

85 patientes	14 mariées	15 - 19 ans	
	42 "	20 - 24 "	
	19 "	25 - 29 "	
	10 "	30 - 34 "	

concluant (Tableau VI). Le maximum se situant entre 20 et 24 ans. La majorité des patientes provenaient de la classe privilégiée.

Septième et Dernier Facteur.—*Localisation*

Nous avons donc remarqué que l'endométriose externe tend à disparaître dans les mariages fertiles à cinq enfants ou plus, mais que la grossesse *per se*, ne semble pas empêcher d'une façon absolue l'endométriose pelvienne, pour ce qui est de l'âge lors du mariage et du temps écoulé depuis le dernier accouchement; il existe une certaine prépondérance de l'endométriose interne chez les multipares et nous avons constaté comme la plupart des auteurs que la localisation la plus fréquente d'endométriose était l'ovaire, 200 cas (84%); Meigs[5] rapporte des séries de 52 à 59%, Fallon 66%, Scott[7] 79%. Notre statistique plus élevée peut être expliquée par l'inclusion des cas d'adénomyose utérine, associés très souvent à l'endométriose ovarienne, et fréquemment diagnostiquée par le chirurgien comme fibrome et endométriose externe. Il demeure difficile de préciser si l'endométriose interne précède ou suit l'externe, et dans quelle mesure, lorsqu'elle la suit, de préciser la cause de la rareté de l'endométriose pelvienne chez les grandes multipares (Tableau VII).

La seconde localisation a été l'utérus (54 cas dont 30 cas d'endométriose mixte, et 24 cas d'endométriose utérine pure) les autres localisations par ordre décroissant de fréquence: 37 cas de Douglas ou de péritoine vésico-utérin, 29 cas des utéro-sacrés, 10 cas de la cloison recto-vaginale, 18 cas de localisation moins fréquente, dont six recto-sigmoïde; viennent enfin les cas de localisation de l'appendice, du vagin, de l'ombilic, du ligament rond, de cicatrices de laparatomie et d'épisiotomie.

TABLEAU VII.—Localisations d'endométriose

238 cas ovaire	200 cas	(84%)
utérus	54 "	(22.7%)
péritoine pelvien	37 "	(15.5%)
utéro-sacrés	29 "	
cloison recto-vaginale	10 "	
autres	18 "	

Conclusion

Nous avons passé en revue dans cette étude les divers facteurs étiologiques pouvant jouer de près ou de loin un certain rôle dans l'endométriose externe. Les deux explications les plus courantes, celle du reflux menstruel avec auto-greffe de l'endomètre dans la cavité pelvienne et la métaplasie de l'épithélium cœlomique, comportent encore des points obscurs. Javert a énoncé une théorie composite comprenant le facteur reflux, associé à des métastases lymphatiques ou sanguines expliquant relativement l'endométriose ganglionnaire et extra-abdominale. Goodall a souligné l'importance des deux modes théoriques de transport à distance de l'endomètre: les couches superficielles de celui-ci sont emportées par le reflux sanguin et étant sensibles aux stimulations hormonales cycliques, ceci donne naissance aux kystes endométriotiques et aux cicatrices étoilées par réaction inflammatoire possiblement secondaire à une irritation par le sang libre; les couches profondes et le stroma endométrial insensibles aux hormones passent par contiguité dans le muscle utérin, puis par métastase lymphatique ou sanguine, vers les ganglions et les localisations atypiques; ces dernières forment l'adénomyose et l'endométriose à tissu plein. Il demeure incontestable que le facteur stérilité joue un rôle important dans l'endométriose mais ce n'est qu'une constatation de faits, sans qu'on puisse expliquer clairement le mécanisme. Il est plus douteux par contre que le facteur grossesse intervienne directement. Les grandes multipares posent un gros problème quant à la diminution de l'incidence du syndrome, mais il subsiste 73 petites multipares qui sont atteintes par la maladie sans que les grossesses n'aient semblé en modifier le cours. Quelles modifications la grossesse, puisqu'elle est si souvent citée comme traitement idéal bien avant les cures hormonales et la chirurgie, quelles modifica-

tions donc, celle-ci apporte-t-elle sur le calibre et le péristaltisme tubaire? Nous ne le savons pas. Quels changements, la présence d'un placenta et la distension considérable de l'utérus durant neuf mois et à répétition, peuvent-elles apporter au point de vue hormonal, morphologique et structural, sur une perméation possible de l'endomètre et dans quelle mesure ces changements sont-ils permanents? La question demeure sans réponse, mais elle est sûrement un point de départ valable d'essai de compréhension de ce syndrome si obscur qu'est l'endométriose.

En conclusion, nous dirons qu'il existe un rapport certain entre la stérilité volontaire ou non, comme cause ou comme effet de l'endométriose; par contre la grossesse peut être considérée sous réserve comme un facteur d'amélioration, à cause de l'incidence assez élevée chez les petites multipares. Comment expliquer aisément alors le cas de la malade de 32 ans, mariée à 16 ans, mère de 10 enfants dont le dernier avait 3 ans?

Résumé

Etude statistique de 238 cas d'endométriose chirurgicale, répartis sur 10 ans. Nous avons réuni à dessein les cas d'endométriose externe et interne.

La plupart des patientes appartiennent à la classe aisée. Le pourcentage des célibataires, femmes mariées, n'est pas significatif.

L'âge moyen était de 30 à 34 ans, avec prédominance des femmes mariées après 34 ans. Nous avons trouvé 25 cas d'endométriose chez les moins de 24 ans. L'adénomyose est fréquente après 35 ans et surtout chez les multipares.

Diminution marquée d'endométriose après cinq grossesses, mais présence d'un bon nombre de cas chez les petites multipares.

L'âge lors du mariage et le temps écoulé depuis le dernier accouchement ne semblent pas jouer un rôle certain.

L'incidence de fertilité a été 57%, la localisation la plus courante étant l'ovaire.

Il y a sûrement une relation stérilité-endométriose, mais la grossesse comme facteur d'amélioration de l'endométriose reste un sujet controversé.

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Summary

This is a survey of 238 cases of surgical endometriosis as observed at the Hôtel-Dieu in Montreal during a period of 10 years. Patients with external endometriosis and those with internal endometriosis have been grouped together intentionally.

Most of the patients were from the higher social strata. There was no significant difference between the percentages of single and married women.

The patients were from 30 to 34 years old. Twenty-five cases of endometriosis were found in patients less than 24 years of age. Adenomyosis was encountered frequently in 35-year-old patients and more especially after multiple pregnancies.

The age at marriage and the length of time which had elapsed since the last pregnancy did not seem to have any influence on the development of this condition. The fertility rate was 57%, and the most frequent localization of the lesion was ovarian. Probably there is an interrelation between sterility and endometriosis, but whether pregnancy improves the clinical course of these cases is still debatable.

THE PROBLEM OF LATE LOCAL RECURRENCE OF CARCINOMA OF THE CERVIX*

J. P. A. LATOUR, M.D. and W. DENNIS FRASER, M.D., *Montreal*

As a result of our perpetual follow-up of gynecological cancer patients at the Royal Victoria Montreal Maternity Hospital since 1926, we are now becoming aware of an increasing number of late recurrences of tumour following what appeared to have been successful treatment of carcinoma of the uterine cervix in terms of "five-year cures".

For the purpose of this report we have excluded those recurrences which appeared within five years of initiaton of treatment. In fact, most of these early recurrences in our experience come within two years of treatment,[2] and for this reason we considered these lesions as a direct progression of the tumour, even if there was an apparent "healing" of the cervical lesion for a short time. We have also excluded patients who died more than five years after treatment, and who, during this long interval, were always found to have tumour present. We are therefore dealing with a group of patients who showed a good initial response to treatment and were considered as "five-year cures".

Definition

It is difficult to be certain that a tumour appearing five years after apparent successful treatment is a recurrence. It could be a new primary lesion of the cervix, a primary carcinoma of the vagina or a tumour induced by radiation.[1] As long as the "recurrent" tumour's histological characteristics conform to the pattern of the primary carcinoma of the cervix, we must admit to the possibility of a recurrence, and it is customary to include all these instances as recurrences.[2]

Site of Recurrence

Recurring tumours of the cervix, whether early or late, are classified according to the site of their recurrence. This may be (a) on the cervix; (b) vaginal wall; (c) paracervical tissues; (d) deposits on pelvic wall; (e) bladder infiltration; (f) rectal infiltration, and/or (g) distant metastases.

Symptoms

The symptoms associated with recurrent tumours of the cervix depend somewhat on the site of the recurrence. Recurrences in the cervix and vagina may cause bleeding per vaginam as well as a purulent discharge. The deeper pelvic recurrences may cause varying degrees of pelvic pain. The pelvic wall deposits tend to cause pain radiating down the thigh, and most characteristically produce a brawny, non-pitting induration of the upper thigh, leaving the lower leg and ankle relatively free of swelling. This "chicken-leg" swelling is one of the classical symptoms of recurrence. Perhaps one of the most important of all symptoms is weight loss, and for this reason our patients are weighed at each follow-up visit. Weight loss is usually present with all types of recurrences and this finding is useful in differentiating parametrial and pelvic wall recurrences from radiation fibrosis, which may present as pelvic pain and swelling of the leg. Pain in the loin may mean obstruction of a ureter by tumour, while oliguria and nitrogenous retention may mean that both ureters are involved.

Using these criteria of selection, we have reviewed our series of 1117 cancers of the cervix treated between 1926 and 1955 inclusive. Follow-up information was available on 1112 cases. There were 44 cases which satisfied our method of selection. These represented 3.9% of the whole series.

These 44 instances of late recurrence included 39 squamous carcinomata and five adenocarcinomata. The latter figure is

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Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Ottawa, January 1961.

TABLE I.—Interval Between Initial Treatment and Recurrence

Years	*No. of cases*
5 - 9	31
10 - 14	7
15 - 19	3
20 - 24	2
25 plus	1

twice the number of adenomatous tumours which one would expect to find in this group. Twenty-three of these 44 cases were histologically proved recurrences, the remainder were included on the basis of indisputable clinical evidence.

Table I shows that most of these late recurrences were between five and 10 years after initial treatment, and that there is a decreasing incidence thereafter. The patient listed as 25 plus years actually went 34 years without symptoms or signs of disease before developing a histologically proved recurrence at the apex of the vaginal vault. This recurrence is much later than those quoted by Truelsen and also is later than Howkins' case which recurred after 30 years.

TABLE II.—Methods of Treatment of the Original Lesion

Original treatment	*No. of cases*
Radium only	22
Radium, x-ray	17
Surgery only	2
Radium, surgery	3

Table II indicates the methods of treatment of the original lesion. It will be noted that a considerable portion of these women received radium alone as definitive treatment. These malignancies date back to the early years of the present series when deep x-ray therapy was not used routinely as adjunctive treatment. When we consider that 322 patients in the entire series were treated by radium alone and 576 were treated by radium plus x-ray, it becomes apparent that the combined treatment is more effective in preventing late recurrence. Of course, this is to be expected because the routine use of deep x-ray therapy has increased the overall five-year survival rate as well.

TABLE III.—Symptoms of Late Recurrent Cancers of the Cervix

Symptoms	*No. of cases*
Pain	15
Bleeding	18
Thigh swelling	10
Weight loss	15

The symptoms presented by these 44 patients with late recurrent cancers of the cervix are listed in Table III. Some patients complained of more than one symptom. Bleeding from the vagina was the most common complaint and was seen in vaginal and cervix recurrences; these cases had a better overall prognosis. Significant weight loss, pain and thigh swelling signified deep-seated malignancy and carried a much poorer prognosis.

TABLE IV.—Frequency of Various Sites of Recurrence

Site of recurrence	*No. of cases*
Cervix	8
Vagina	14
Parametria	18
Pelvic wall	12
Bladder	7
Rectum	2
Distant	13

The location of the recurrence was established in most cases on clinical grounds with judicious use of other diagnostic aids. The frequency of the various sites of recurrence is shown in Table IV. It will be noted that the number of sites exceeds the number of patients because several patients had more than one area of involvement.

An association between the age of the patient at the time of onset of the original tumour and the frequency of late recurrence was sought, but we were not able to demonstrate any correlation in this direction. However, when the stage of the original lesion was examined it was found that 22 of our late recurrences were originally Stage I, 17 were Stage II, five were Stage III, and none were Stage IV. This paradox is readily explained on the basis of the greater rate of five-year survival in the earlier stages, but since there were no five-year survivors in Stage IV there were no opportunities for late recurrences.

TABLE V.—Types of Treatment in Recurrent Cases

Type of treatment	*No. of cases*
No treatment	8
One course of deep x-ray	12
Two courses of deep x-ray	6
Local radium	6
Cobalt^{60}	4
Local excision	2
Chemotherapy	1
Colostomy	3
Colpectomy	1
Colpocleisis	2
Uretero ileocystoplasty	3
Anterior exenteration	2

The clinical management of these patients with late recurrent carcinoma of the cervix varied widely according to the condition of the patient. Table V shows that a number of patients were given no specific treatment beyond symptomatic relief of pain and other symptoms. Local radium treatment was applied in a few cases where bleeding was the chief complaint. The most common method of treatment was external radiation, either as deep x-ray or Cobalt^{60}, and frequently more than one course of such therapy was given. The variety of surgical procedures carried out were chiefly to correct ureteric and rectal obstruction or leakage. In two cases, a more extensive surgical procedure was carried out with a view toward achieving more lasting results. Chemotherapy was used once in this group of patients.

TABLE VI.—Survival Time after Treatment of Recurrence

Time interval	*Patients dead*	*Patients still alive*
Less than 1 year	13	2
1 to 2 years	13	1
2 to 5 years	10	2
5 to 10 years	—	2
Over 10 years	1	—

The results of treatment were disappointing but were not entirely without some gratification. Table VI indicates that many of these patients died within a year of their recurrence and the majority were dead within two years. However, there were a few who survived beyond two years, and three of these have survived their recurrences by more than five years. One patient survived a proved recurrence for 17 years to die suddenly of a heart attack.

Conclusions

In conclusion it appears that the perpetual follow-up of cancer patients will reveal a considerable number of late recurrences, some of which may appear many years after an apparent "cure". It is evident that five-year or even 10-year survival figures do not take late recurrences into account. Recurrent tumour of the cervix is not easy to identify unless it involves vagina or cervix. The majority of the recurrences are confined to the pelvic cavity and although a reasonably accurate diagnosis can be made eventually, the early signs and symptoms may be subtle and cause delays in the appreciation of the true nature of the trouble. Therefore a careful check for the symptoms already listed is recommended.

Once diagnosed, the recurrence is frequently amenable to some therapeutic control and the results obtained are somewhat encouraging.

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Résumé

Après avoir suivi pendant longtemps les malades traitées pour cancer du col utérin, les auteurs se sont aperçus qu'il existe un pourcentage important de récidives chez des patientes qui avaient été considérées comme guéries "après cinq ans". Ils ont groupé ces cas en excluant ceux de moins de cinq ans.

Il est parfois difficile de savoir si une tumeur qui apparaît cinq ans après une autre tumeur est réellement une récidive; cela pourrait être en effet une deuxième tumeur primaire ou une tumeur provoquée par les radiations. Les auteurs ont donc décidé de classer comme récurrence toute tumeur dont l'histologie était celle d'un épithélioma du col.

La localisation de ces récidives varie. On en trouve sur le col utérin, sur la paroi vaginale, dans les tissus paracervicaux, dans la paroi du bassin, dans la paroi vésicale, dans le rectum et enfin comme métastases lointaines. Cette variation dans la localisation influe évidemment sur la symptomatologie.

On a ainsi trouvé pour une période de 1926 à 1955, 44 cas de récidives tardives, dont 39

carcinomes squameux et 4 adénocarcinomes. Différentes considérations relatives à ces cas sont apportées sous formes de tableaux. On constate notamment que les récidives semblent avoir été moins importantes chez les malades traitées par le radium et les rayons X.

En conclusion, les auteurs attirent l'attention sur le fait que les récidives tardives du cancer du col utérin existent: c'est pourquoi il est important de suivre les malades plus longtemps que cinq ans et de guetter tout symptôme capable de suggérer le cancer.

HYPERTENSIVE REACTION FOLLOWING RESECTION OF COARCTATION OF THE AORTA

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SURGICAL correction of coarctation of the aorta is now a standard procedure. The clinical and technical aspects of the condition have been well documented.[1] Although resection of the coarctation is nearly always followed by prompt relief of the hypertension, occasionally a transient increase in blood pressure occurs. The mechanism of such a further elevation is speculative. This report concerns our observations on four patients who developed paradoxical hypertension after surgical correction of their coarctation.

CLINICAL MATERIAL

Eleven patients between four and 30 years of age underwent operation for coarctation of the aorta between 1957 and 1960. Five patients in this group presented with symptoms referrable to coarctation. Femoral pulses were palpable in four and questionable in one, but systolic and diastolic blood pressure was raised in every patient. All were the post-ductal types and in three patients the ductus was patent. A satisfactory result was obtained by end-to-end anastomosis and concomitant ligation of the ductus when present.

Heart rate, cuff blood pressure in the arm and thigh, and digital pulse volume and blood flow in the lower extremities were recorded preoperatively. Postoperatively, heart rate and blood pressure readings were obtained four times daily until discharge. Digital pulse volume and blood flow in the extremities were recorded daily for five days and every other day thereafter for the next 10 days.

Pulse volume and blood flow were obtained in the following manner. A plethysmograph was attached to the second toe and rendered air-tight by the use of "stripseal putty". A blood-pressure cuff was placed at the ankle so that the blood flow could be measured by the venous occlusion method. A Grass plethysmograph was the recording apparatus in this study.

POSTOPERATIVE COURSE

In seven patients the systolic and diastolic blood pressure fell appreciably after operation, the average fall in the systolic blood pressure being 28 mm. Hg. In every case the femoral pressure exceeded the arm pressure.

The postoperative course was complicated in four patients by paradoxical hypertension which was alarming (Table I). In a boy of 16, a systolic rise of 40 mm. Hg over the preoperative level developed within three hours of operation. In the other three patients with hypertensive crises, an average systolic rise of 25 mm. Hg over the preoperative level occurred within 12 to 48 hours after operation. These four patients were treated with ganglionic blocking agents and barbiturates for 10 to 30 days.

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TABLE I.—Paradoxical Postoperative Hypertension

	Preoperative blood pressure						*Two weeks*	
	Arm	*Thigh*	*1st day*	*2nd day*	*5th day*	*10th day*	*Arm*	*Thigh*
1. W.S.	180/130	105/125	220/150	230/140	220/130	200/130	170/120	190 systolic
2. L.H.	160/80	Faintly palpable	180/95	200/130	190/120	180/100	170/90	185 systolic
3. B.W.	130/65	Not recorded palpable	190/85	170/80	140/65	140/65	135/70	150 systolic
4. N.M.	130/80	100/85	170/90	165/90	145/85	140/80	135/80	150 systolic

A marked tachycardia of 110 to 130 per minute occurred in three patients and lasted from 12 to 16 days. One of these patients, a girl of four, also showed paradoxical hypertension 190/85 mm. Hg about 12 hours after correction of the coarctation of the aorta, the initial blood pressure being 130/85 mm. Hg (Fig. 1).

Digital pulse volume and blood flow studies in the lower extremity showed in each instance a 400 to 600% increase over the preoperative levels (Fig. 2). In the group of patients showing paradoxical hypertension, increases in blood flows were of the same order (Fig. 3). There was no evidence of vasoconstriction in the measurements made by digital plethysmography.

Discussion

Severe hypertension occurring after successful repair of coarctation of the aorta may lead to disruption of the suture line. In each of our four patients there were no ill effects as a result of this complication. March, Hultgren and Gerbode[2] recently reviewed 80 patients, of whom three died within three to five days, and in two others death occurred in two to six weeks after operation. Death was due to aortic rupture and hemorrhage secondary to postoperative hypertension.

In the literature two distinct types of postoperative hypertension have been described as "early" and "delayed", the latter occurring after the second postoperative day. The delayed type is often associated

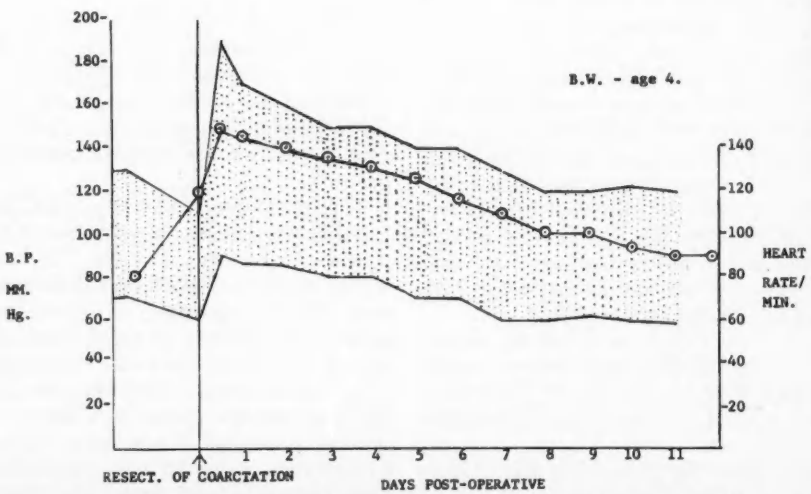


Fig. 1.—Patient B.W. Graph showing blood pressures (stippled area) and heart rate (θ —— θ) over the 11 days following resection of the coarctation of the aorta.

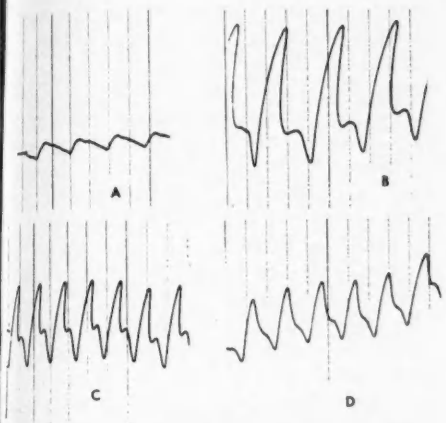


Fig. 2.—Patient W.S. Pulse volume tracings. A, B, C and D represent preoperative, fourth postoperative day, tenth postoperative day and six weeks postoperative.

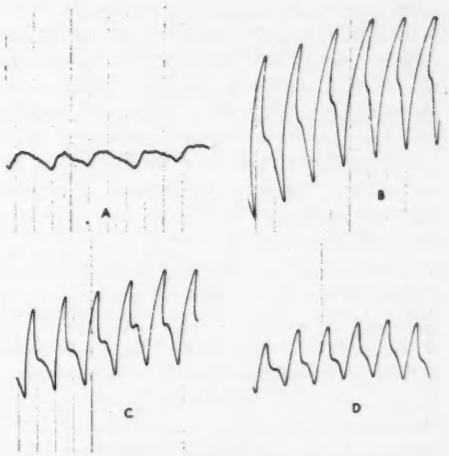


Fig. 3.—Patient B.M. Pulse volume tracings. A, B, C and D represent preoperative, fourth postoperative day, tenth postoperative day and six weeks postoperative.

with abdominal pain and distension according to Sealy *et al.*,[3] who reviewed 30 patients. In their group, seven patients had early hypertension, and delayed hypertension developed in 14. Six patients in the latter group developed abdominal pain and distension and in two patients laparotomy showed segmental bowel necrosis. The association of paradoxical hypertension and necrotizing arteritis in the abdominal organs has been documented.[4, 5]

The incidence of postoperative hypertension in the review of March *et al.*[2] was "early hypertension" in eight (14%) and "delayed" in 31 (53%). These authors did not find a true correlation between postoperative hypertension and necrotizing arteritis. The high incidence of paradoxical hypertension, both early and delayed in the two reports found in the available English literature and as shown in our small group of patients, indicates that this is a frequent occurrence.

What then is the mechanism of this paradoxical hypertension which occurs so frequently after an operation carried out to cure hypertension by relieving a mechanical obstruction to blood flow? The second question is why do some patients develop hypertension early and others two to three days after operation? In the present state of our knowledge the answer to these questions can only be surmised. Is the hypertension renal in origin, subsequent to multiple emboli? The patients in Sealy's series who were examined postmortem showed no renal pathology.

Postoperative hypertension can occur if severe narrowing occurs at the site of anastomosis due to technical difficulties. d'Abreu[6] reports two such cases. In our group of patients the femoral cuff pressure postoperatively in each case was higher than the arm cuff pressure, so that postoperative hypertension cannot be explained on this basis.

In some of the reported cases paradoxical hypertension has been associated with necrotizing arteritis in the lower half of the body. Is this hypertension associated with vasoconstriction in the lower half of the body? This could be induced by the sudden increase in the pressure head subsequent to removal of the obstruction in the aorta. With this possibility in mind, blood flows to the lower extremity were studied. These studies showed that blood flow to the toes was increased 400 to 600% over the preoperative level. There is no evidence of vasoconstriction at least in the cutaneous bed.

It appears reasonable to accept the hypothesis of Sealy that the immediate type of hypertension is due to the activity of the

aortic and carotid pressure receptors which were set at a high level before operation. Subsequent to resection of the coarctation, the tension in the aortic arch and carotid arteries falls. Thus the aortic and carotid pressure receptors after operation allow a rise in blood pressure by failing to act as buffers. This hypothesis fails to explain the hypertension occurring two to three days after operation.

After resection of a coarctation the blood supply to the lower half of the body is increased; the adrenals share this increased nourishment. It is possible that epinephrin and norepinephrin secretion is increased, giving rise to hypertension. Urinary catecholamine estimations before and after resection of coarctation will be of some value. Preliminary estimations, however, in several of our patients showed no alteration, although other authors[7] have shown a correlation between the elevated pressure and an elevation of urinary catecholamines.

Thus, from our investigations, there is no acceptable explanation for the serious temporary hypertension which may follow resection of a coarctation of the aorta. Such hypertension must be due to either (a) increased cardiac output; (b) increased peripheral resistance; (c) increased blood volume; (d) decrease in the total volume of the vascular bed, or (e) to a combination of any two or more of these variables.

The plethysmographic studies in our patients indicate that there is no increase in peripheral resistance in the lower limb at least. We have no knowledge of the peripheral resistance in the upper extremities or in the splanchnic bed. If there were an excessive secretion of catechol pressor amines, secondary to increased blood flow to the adrenal glands, we would expect evidence of excessive vasoconstriction of digital vessels of the lower extremities. Again, if the carotid and aortic pressor receptors are set at a high level, why do they not come into action when the severe hypertensive phase is present in order to cause an immediate lowering of blood pressure? We are faced with the paradox of apparent marked vasodilation below the level of the resected coarctation, and a hypertension which should be associated with a marked constriction of this major portion of the vascular bed. A sudden expansion of blood volume is hard to imagine. We would rather expect the reverse to be true because of the increased filtration force in the lower half of the body secondary to the resection of the coarctation. A rapid diminution in the total volume of the vascular bed is again hard to visualize, in so far as this is not borne out by the studies on the lower limbs in which there would appear to be an expansion of the vascular bed. Any marked increase in cardiac output following the resection of a coarctation has not been investigated, but is a distinct possibility. This increase would have to be out of all proportion to the volume of the vascular bed.

Thus this preliminary communication indicates that, following resection of coarctation of the aorta, there is a major disorganization of the homeostatic mechanisms which regulate the hemodynamics of the cardiovascular system. With removal of the narrowed area of the aorta, the full force of the pulsatile blood flow under full systemic pressure is transmitted into a large part of the vascular bed, which has not been accustomed to sustaining such forces. Why then does the vascular system respond by excessive systemic hypertension? Obviously, in order to solve this problem, there is a need for investigation of the post-resection state of the blood volume, of the total peripheral resistance, of the condition of the splanchnic bed, of the condition of the vascular bed of the upper half of the body and of the cardiac output.

In conclusion, alarming hypertension frequently follows resection of coarctation of the aorta. The explanations for this can only be surmised. The effect of such increased pressure on the anastomosis can be fatal rupture if the pressure is not vigorously controlled with barbiturates and antihypertensives.

Summary

Eleven patients with coarctation of the aorta were treated by resection of the stenotic segment and end-to-end anastomosis without mortality.

In four, the postoperative course was

complicated by paradoxical hypertension. All four were successfully treated with barbiturates and antihypertensive drugs.

The peripheral circulation was studied before and after resection of coarctation by measuring blood flow using a digital plethysmograph. All patients, including the ones with paradoxical hypertension, showed a 400 to 600% increase in the blood flow to the toes.

The possible explanations for this phenomenon are briefly discussed, and the unanswered questions have been put forward.

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Résumé

La tactique chirurgicale pour la coarctation de l'aorte est maintenant bien au point. Bien que, dans la plupart des cas, cette intervention soit suivie d'une prompte cédation de l'hypertension, il arrive cependant qu'une hypertension paroxystique s'installe à titre transitoire. Le mécanisme de ce phénomène reste obscur.

Dans une période de trois ans, 11 patients dont les âges variaient entre quatre et 30 ans furent opérés pour cette affection. Sur ce nombre, quatre eurent des suites opératoires compliquées d'hypertension paroxystique.

L'hypertension qui s'installe dans ces conditions est inquiétante car elle peut provoquer une rupture de la ligne de suture. Ces crises d'hypertension peuvent survenir soit précocement, soit tardivement (plus de deux jours post-opératoires). Une hypothèse intéressante permettant peut-être d'expliquer l'hypertension précoce est la suivante: les récepteurs tensionnels aortique et carotidien se trouvent, avant l'opération, travailler à un haut niveau. Après l'intervention, la tension sanguine diminue dans l'arc aortique et les carotides, de sorte que les tenso-récepteurs provoquent une hypertension réactionnelle.

Il ne semble pas exister d'explication très valable de l'hypertension tardive. Les auteurs discutent ici ce point en détail et communiquent diverses observations personnelles.

MEASUREMENT OF ARTERIAL FLOW IN THE LOWER EXTREMITIES WITH RADIOISOTOPES*

"Radioisotopes have previously been used in attempts to determine the rate of blood flow in extremities. Their use was provoked by general dissatisfaction with the older methods of oscillometry, plethysmography, and colorimetry.

"Radioactive sodium and I^{131}-labelled serum albumin have been the agents chiefly employed. Scintillation counters have proved most satisfactory as detection devices. Two general methods have been used; first, determination of the build-up of activity in a region after intravenous injection, and second, measurement of the rate of removal of a labelled diffusable local injection into the tissue. Scintillation counters placed regionally are then used to determine the rate of build-up, or 'wash-out', of the substances . . .

"The shortcomings of the methods mentioned with their apparent inaccuracies and procedural complexity have relegated them to the research laboratory. There has been a need for a relatively simple, accurate, and objective method of evaluating patients with claudication-like symptoms. We have developed a method which satisfies these prerequisites. The method has been effectively used as a screening device and as an adjunct to arteriography . . .

"A method in which radioactive iodinated serum albumin is utilized for the evaluation of the rate of blood flow in extremities has been presented. The reliability of the method was tested in the laboratory on animals and substantiated by mathematical analysis. The efficiency of the method is undeniable and it is safe. The dosage of the tracer used is less than that used for a thyroid uptake test."

*Applen, J E., Lerman, G. S. and Walker, P. E.: *Surgery*, **50:** 315, 1961.

SURGICAL EXPERIENCE IN RESECTION OF ANEURYSMS OF THE THORACIC AORTA*

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CHIEFLY UNDER the guidance and stimulus of Dr. Michael De Bakey, surgery of the aorta has progressed to its present state of high achievement, advancing in the past ten years from a "smash-and-grab" technique to one of increasing perfection.

In 1959 we became interested in aneurysms of the thoracic aorta and the possibility of their resection with the help of surgical bypass techniques. The result of our two-year experience has been the surgical treatment of 11 patients, nine males and two females ranging in age from 52 to 74 years, with the exception of two patients who were 22 and 28 years of age. Etiologically, six of these aneurysms were due to arteriosclerosis, three to syphilis, one to trauma and one developed on the basis of idiopathic cystic medial necrosis of the ascending aorta.[1]

DIAGNOSIS

The presenting symptom in nine patients was chest pain which led to medical consultation, while two lesions which were asymptomatic were found on routine chest radiograph. Two patients had sharp intermittent chest pain of segmental type from pressure on an intercostal nerve trunk, while in seven the pain was deep-seated, constant and poorly localized to the anterior or posterior part of the left chest. However, in the majority of patients the important features of the chest pain were chronicity, lack of relation to exercise and relief with mild sedation. Indeed, when a patient presents with this pain pattern, one should include aneurysms of the thoracic aorta in the differential diagnosis. Yet, in five patients, the pain was misinterpreted and serious delay in surgical treatment resulted. In one patient, arthritis of the dorsal spine was thought to be the source of pain, and in two others, coronary artery occlusion was diagnosed in spite of repeated normal electrocardiograms. Moreover, in one of the patients, anticoagulant therapy was associated with rapid expansion of the aneurysm. The two patients with intercostal neuritis were treated on the basis of idiopathic non-specific inflammation. The common factor in these five patients was the great loss of time with medical therapy before surgical intervention.

Radiographs and fluoroscopy are extremely helpful in differentiating thoracic aortic aneurysms from solid tumours of the mediastinum and hilar region of the lung. However, one must keep in mind that on fluoroscopy many aneurysms do not pulsate because of thick laminated thrombus within the wall, or because the wall is thin and under high pressure. Conversely, many solid tumours of the mediastinum will transmit pulsations from the great vessels. To overcome this diagnostic error and also to determine the extent of thoracic aneurysms, retrograde aortograms are performed through the right brachial or femoral artery in all our cases when surgery is contemplated.

The two cases which follow illustrate both aspects of this diagnostic problem.

CASE 1.—Six weeks before admission this 71-year-old man developed vague left upper chest pain, which radiated to the posterior chest. Driving a car increased the pain and mild sedation relieved it. The patient was otherwise well. Physical examination was negative. A radiograph of the chest showed a round, smooth 12-cm. mass in the left superior mediastinum, which depressed the aortic arch, but did not appear to be attached to it (Fig. 1). The trachea was displaced 2½ cm. to the right. On fluoroscopy, the mass did not pulsate and was diagnosed as a solid or cystic tumour of the superior mediastinum. At surgery, the mass was discovered to be a tense, thin-walled aneurysm of the aortic arch, which could have been diagnosed preoperatively if an aortogram had been performed.

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Presented at the Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Ottawa, January 1961.

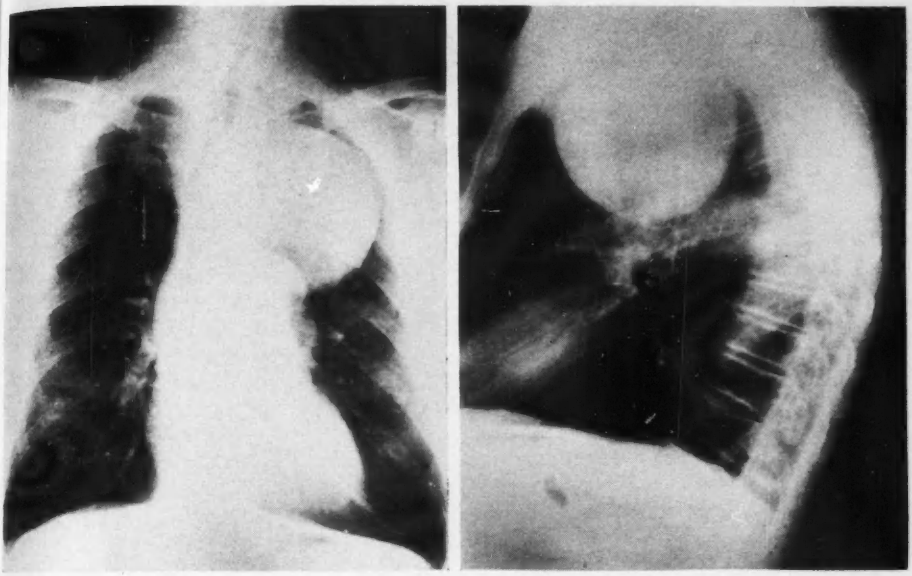


Fig. 1.—The mass depresses aortic arch but does not appear to be attached to it.

CASE 2.—A 62-year-old man noted increasing hoarseness for two months, associated with vague pain in the anterior left chest, which was present most of the time and was relieved by phenacetin. There were no abnormal auscultatory findings, but direct laryngoscopy showed that the left vocal cord was paralyzed. A radiograph of the chest was interpreted as showing enlargement of the aortic arch and on fluoroscopy the mass pulsated (Fig. 2). The

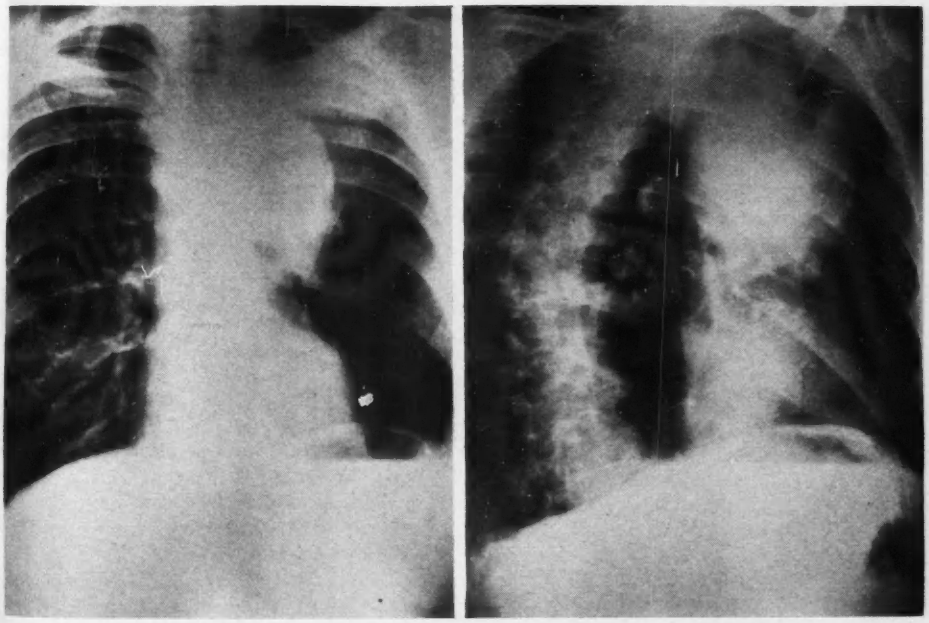


Fig. 2.—The mass pulsated on fluoroscopy, but aortogram was normal.

mass was believed to be a thoracic aneurysm until aortography performed through the right brachial artery showed a normal thoracic aorta. The patient was subsequently given radiation therapy for inoperable anaplastic carcinoma situated in the hilum of the left lung.

Technical Aspects

Since surgical correction of coarctation of the aorta was introduced 15 years ago, surgeons have continued to devise new procedures to deal with obliterative and hemodynamic lesions of the thoracic aorta. Ample collateral circulation around a coarctation permits cross-clamping of the aorta, proximal and distal to the lesion with relative disregard of time. However, the absence of collateral circulation about most thoracic aneurysms renders cross-clamping of the aorta extremely hazardous unless the patient is supported during the procedure with surgical bypass techniques.[2]

If the thoracic aorta is cross-clamped, hypertension develops in the proximal segment and profound hypotension in the segment distal to the clamp. Hypertension may result in cerebral hemorrhage or left ventricular failure from acute distension of that chamber. Hypotension in the distal segment of the aorta may produce irreparable spinal cord damage because the chief components of the anterior spinal artery are the radicular branches of the thoracic aorta. This is true particularly if the aorta is cross-clamped proximal to the seventh dorsal vertebra.

Adaptation of the Sigma motor pump,* with or without an oxygenator, to this type of surgery has prevented the development of a serious pressure gradient in the cross-clamped aorta by removing blood from the left atrium and pumping it through the femoral artery into the aorta distal to the occluding clamp. Adequate femoral artery perfusion preserves and maintains the function of the abdominal organs. Moreover, this form of bypass prevents serious left ventricular strain by maintaining blood pressure at approximately normal levels. No spinal cord damage occurred in our eight cases in which the left atrial pump bypass was used. De Bakey reported two cord lesions in 55 patients in whom only temporary grafts were used to bypass the area of aortic occlusion.[3] It would appear that variation in the segmental distribution of the spinal artery and lack of development of collateral circulation were responsible for these lesions. Before the left atrial-femoral artery bypass technique could be used, it was necessary to find ways of regulating the blood pressure, of controlling the perfusion rate and maintaining a stable circulatory volume.

In dealing with aneurysms of the descending aorta, the lesion is approached through a left thoracotomy. Tapes are placed proximal and distal to the aneurysm for control, and a plastic cannula, inserted into the left atrium through the appendage, is connected to a venous well (Fig. 3). Blood passes to the Sigma motor pump and into the common femoral artery. Arterial pressure proximal to the lesion is monitored through the left internal mammary or radial artery, while pressure distal to the aneurysm is recorded through a catheter in a common femoral artery. The patient is heparinized and, immediately after the aorta is clamped above and below the aneurysm, the pump is set to draw 1500 c.c. per minute from the left atrium. Changes in flow are regulated by the proximal and distal pressure recordings; if the area proximal to an aneurysm becomes hypertensive, the flow from the left atrium is increased until equal pressures are restored. Blood lost by suction is measured and constantly replaced to maintain a fixed circulating volume.

Once the aneurysm is replaced by a synthetic graft, the aortic clamps are removed and the pump is stopped. Usually, the left ventricle withstands the sudden hemodynamic change without difficulty, in spite of the older age of most of the patients. Since the success of this technique depends on the left ventricle perfusing the patient proximal to the occluding clamps, any factor such as hypothermia, which increases ventricular irritability, particularly in older patients, is avoided when employing the left atrial-femoral artery technique.

Aneurysms of the aortic arch proximal to

*Sigma Motor Pumps, Middleport, New York.

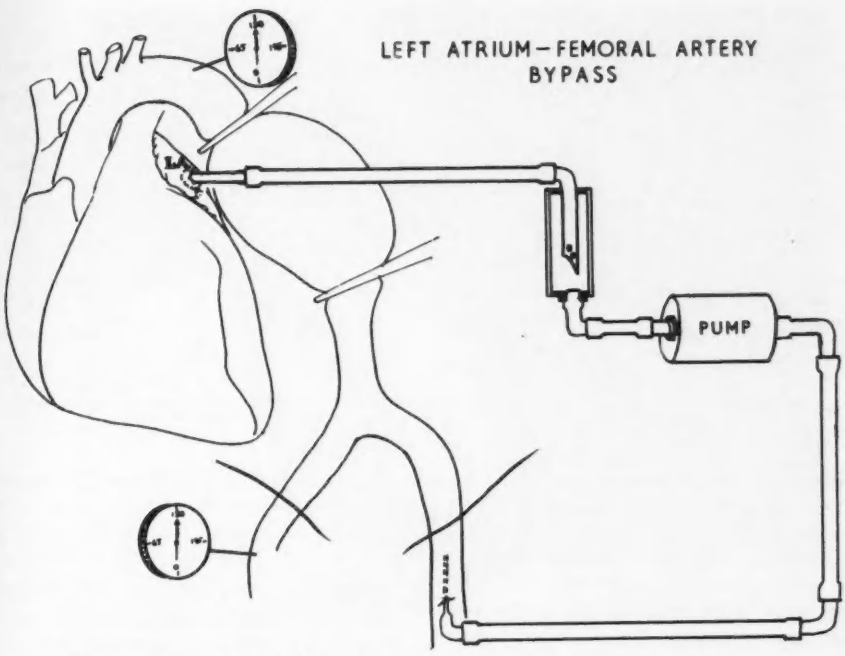


Fig. 3.—Diagram of left atrial-femoral artery bypass technique. A heat exchanger can be added to the arterial line.

the left subclavian artery pose a more difficult problem than those of the descending aorta. Indeed, cross-clamping of the ascending aorta for several minutes results in left ventricular failure and irreversible cerebral ischemia. Consequently, the choice of a surgical technique in aneurysms of the ascending aorta and arch is decided upon when the surgeon sees whether or not the proximal half of the ascending aorta contains a fusiform aneurysm. If the proximal half of the ascending aorta is not involved, the same technique as used for aneurysms of the descending aorta is chosen, with the addition of perfusion cannulae inserted into the innominate and left common carotid arteries. The aortic arch is resected and a synthetic graft inserted. With this technique the ascending aorta is cross-clamped proximal to, and the descending aorta distal to the aneurysm. The cannulae to the carotid arteries are extensions from tubing connecting the left atrium to the venous well (Fig. 3). The end-to-end teflon graft is inserted, with openings for anastomosis with the innominate and left common carotid arteries. The left subclavian artery is not usually anastomosed to the graft. On completion of the anastomoses, the two small plastic cannulae are removed from the carotid arteries and the small side openings repaired with 5-0 silk sutures.

If, however, the proximal half of the ascending aorta contains a resectable fusiform aneurysm, the pump oxygenator and cardiac hypothermia are used (Case 11).[4] The aneurysm is approached through a median sternotomy, a single large catheter is inserted into the right ventricle via the atrium and a second catheter is placed in the left atrium for decompression of the left heart during induced cardiac hypothermia. Blood from the pump oxygenator is returned to the patient through the common femoral artery. The ascending aorta is cross-clamped and divided between the aneurysm and the innominate artery. Cold blood is perfused into the clamped segment of the ascending aorta to induce cardiac arrest, while the distal anastomosis with a synthetic graft is performed. The

TABLE I.

Name	*Age*	*Etiology*	*Type and extent*	*Treatment*	*Result*
1. D.P.	58	Syphilis	Saccular—ascending aorta	Ruptured during clamping of neck	Died during surgery
2. A.B.	69	Arteriosclerosis	Saccular—descending aorta	Resection and graft, left atrial-femoral artery bypass and hypothermia	Died 3 hours after operation—ventricular fibrillation
3. D.C.	52	Arteriosclerosis	Fusiform—descending aorta	Resection and graft, left atrium-femoral artery bypass	Well at 20 months
4. Mrs. M.B.	57	Syphilis	Fusiform—descending aorta and left half of arch	Resection and graft, left atrial-femoral artery bypass	Well at 19 months
5. J.W.	74	Syphilis	Fusiform—ascending arch and descending aorta	Thoracotomy—lesion inoperable—Ivalon wrapping	Arch ruptured on 4th day—patient died
6. M.J.	68	Arteriosclerosis	Fusiform—descending aorta	Resection and graft—left atrial-femoral artery bypass	Well at 15 months
7. W.M.	70	Arteriosclerosis	Saccular—descending aorta	Resection and patch graft left atrial-femoral artery bypass	Well at 12 months
8. H.B.	67	Arteriosclerosis	Fusiform—distal half ascending aorta and arch	Resection and graft left atrial-femoral artery bypass	Died of acute left ventricular failure during bypass procedure
9. V.I.	28	Trauma	False aneurysm junction arch and descending aorta	Resection and graft left atrial-femoral artery bypass	Died on 7th day of staphylococcal pneumonia
10. Mrs. C.S.	67	Arteriosclerosis	Fusiform—descending aorta	Left atrial-femoral artery bypass	Died of ventricular fibrillation during bypass procedure
11. J.J.	22	Cystic medial necrosis	Fusiform—ascending aorta	Resection and graft, pump oxygenator and cardiac hypothermia	Well at 2 months

ascending aorta is then resected just distal to the coronary ostia, while cardiac hypothermia is maintained by intermittent perfusion of the coronary arteries till the proximal anastomosis is completed. The heart is resuscitated by removing the aortic cross-clamp and perfusing the coronary arteries with normothermic blood from the pump oxygenator.

Discussion

In our series of 11 patients, five are well and active 2 to 22 months after surgical resection of the thoracic aneurysm (Table I). Two patients survived operation but died in the postoperative period, one (Case 2) of acute coronary artery occlusion and the other (Case 9) one week after surgery, from extensive staphylococcal pneumonia. Patients 8 and 10 died of acute left ventricular failure following several minutes of hypotension during left atrial-femoral artery bypass. In using this technique, adequate function of the left ventricle is essential to perfuse the brain and to supply blood for the venous well used in perfusion of the distal aortic segment. Consequently, it is important to maintain normal pressure in the proximal aortic section to ensure adequate coronary artery perfusion and left ventricular function; conversely, if the pressure is too high because of hypervolemia, the left ventricle may not tolerate the extra work and dilatation and fibrillation may result.

Cases 8 and 10 emphasize the necessity of constant pressure control in the arterial system proximal to the aortic clamp, particularly for those patients who have arteriosclerotic heart disease and withstand even transitory hypotension poorly because it reduces coronary perfusion and predisposes to ventricular fibrillation. It is advisable to add calcium chloride in the amount of 0.5 to 1 g. periodically into the pump circuit when myocardial tone is poor or when significant cardiac arrhythmia develops. Moreover, for the same reason general or cardiac hypothermia should be avoided unless an oxygenator is used with the pump as in resections of aneurysms of the ascending aorta (Case 11).

We prefer woven teflon grafts because their reduced porosity permits rapid hemostasis and avoids the necessity of preclotting before its use (Fig. 4). Furthermore, teflon can be fashioned quickly and segments can be added easily for aortic arch vessels.

Summary

The choice of elective surgery with thoracic aneurysms is based upon the estimation that the patient has at least five years of active life remaining. Emergency operation upon thoracic aneurysms is rare compared with those in the abdomen; in our series this was carried out in only one



Fig. 4.—Completed teflon graft of descending aorta. Ivalon cuff used to reinforce distal anastomosis.

patient (Case 2). Rupture of thoracic aneurysms is usually precipitous; emergency operation is extremely hazardous and carries a forbidding mortality. For this reason, excision of the aneurysm upon diagnosis should be the procedure of choice in centres where this type of vascular surgery is well established.

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Résumé

Les auteurs présentent ici leurs expériences dans le traitement chirurgical des anévrysmes de l'aorte thoracique portant sur 11 cas, dont neuf hommes et deux femmes, d'âges variant entre 52 et 74 ans.

Dans la plupart de ces cas, le symptôme principal était une douleur dans le thorax qui amena à consulter; les anévrysmes furent découverts alors par la radioscopie. Cette douleur était chronique et sans aucune relation avec l'exercice.

L'examen radiologique est évidemment très important; il faut se souvenir cependant que, à la fluoroscopie, les anévrysmes aortiques ne sont pas toujours pulsatiles. De plus, certaines tumeurs peuvent sembler pulsatiles simplement parce qu'elles transmettent les pulsations des gros vaisseaux. Le diagnostic différentiel devra donc être fait par aortographie rétrograde.

De cette série de 11 malades, deux moururent dans la période post-opératoire, l'un d'une occlusion coronaire et l'autre d'une pneumonie à staphylocoques. Deux autres malades succombèrent à la suite d'une défaillance ventriculaire gauche. Un mourut d'une rupture de l'arc aortique au quatrième jour. Un enfin succomba pendant l'opération, l'anévrysme s'étant rupturé à sa base lors de l'essai de clampage. Cinq patients sont actuellement en très bonne santé.

THORACIC SEQUESTRATION CYSTS OF FETAL BRONCHOGENIC AND ESOPHAGEAL ORIGIN*

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THORACIC cysts of fetal bronchogenic origin usually do not cause symptoms until adult life,[1] and their surgical removal is not difficult. In contrast esophageal sequestration cysts, which show some histological similarities, are dissimilar and tend to produce symptoms in infancy because of their position. Furthermore, they are quite difficult to resect. These cysts have been confused especially on histological grounds and are described under a plethora of names which obscures their usual surgical characteristics, and even merges them with other entities.

Up to 1946 there were only 35 case reports of bronchogenic cyst in the literature.[2] After the introduction of photofluorographic mass radiography surveys this apparent rarity was rare no longer; for example, in the same year 23 more instances were diagnosed by the American Army Thoracic Surgical Centers alone.[3] We wish to report three bronchogenic lung-bud and two esophageal sequestration cysts in order to compare and contrast the features of each.

Bronchogenic Cysts

CASE 1.—A previously healthy woman first developed recurrent attacks of paroxysmal dry cough, with fever ranging up to 102° F., at 48 years of age and with these symptoms she noted a sensation of aching deep in her right chest. Physical examination was negative, but chest roentgenograms showed a coin lesion lateral to the right hilum and immediately above the lower portion of the major fissure (Figs. 1 and 2). No peripheral atelectasis, notching or lobulation of outline was detected to suggest bronchogenic carcinoma. No tumour cells or tubercle bacilli were seen on sputum examination. Bronchoscopy and bronchography showed no significant change.

The mass increased slightly in size on radiological examination when she was sick, shrinking again when asymptomatic. At thoracotomy a semi-translucent cyst, 3½ cm. in diameter, was found in the right middle lobe, abutting on hilar structures and enclosed within a thin connective tissue capsule. This capsule was dissected free without difficulty, and no bronchial communication or aberrant vessels were encountered. Microscopically the bronchogenic cyst showed a typical inner lining of ciliated pseudostratified columnar epithelium lying on a thin connective tissue wall containing a very thin plate of bronchial cartilage. No glands, muscle or thickened basement membranes were detected. The patient has remained healthy during a two-year follow-up study.

CASE 2.—This male patient was first seen at the age of 76 years with a right inguinal hernia of recent origin. This was aggravated by chronic dry cough of many years standing which had never responded to antibiotic treatment. Chest roentgenograms (Figs. 3 and 4) showed a sharply circumscribed homogeneous coin lesion, 4½ cm. in diameter, at the level of the aortic arch. Herniorrhaphy was carried out, and on follow-up over the next three years no change was observed in appearance of the lung lesion. However, the cough became more productive and he noted that coughing was provoked especially by movement.

He was injured in a car accident and readmitted to hospital with multiple fractured ribs, and died 24 hours later from the effects of left pneumothorax and terminal bilateral bronchopneumonia. At autopsy the lower two-thirds of the left upper lobe was occupied by a round paper-thin cyst, 7 cm. in maximum diameter. Its inner lining was smooth and pale and enclosed a soft but solid mass of greasy yellowish-brown debris. No communication with the bronchus was demonstrated and no scarring detected in the adjacent lung parenchyma, which showed minimal compression. Microscopically the contents were meshed hyphal filaments of an aspergilloma or "fungus ball". The cyst lining was formed by ciliated respiratory epithelium, with occasional bronchial glands, a small amount of plain

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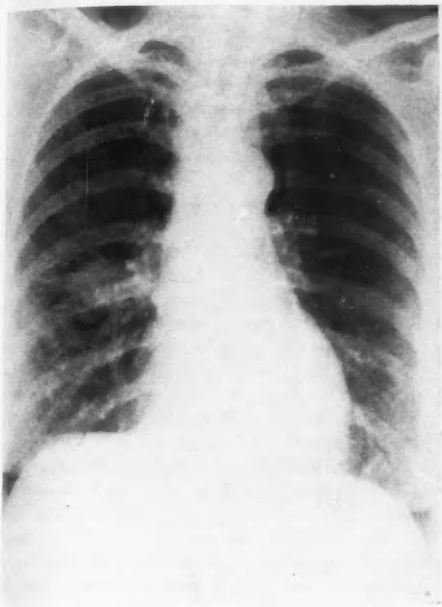


Fig. 1

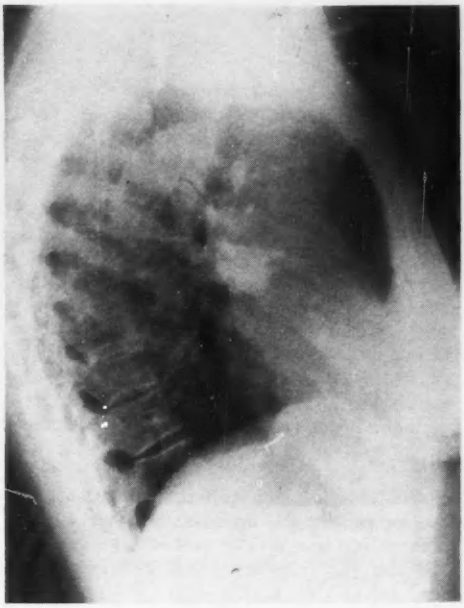


Fig. 2

Fig. 1.—Case 1. A large coin lesion lies adjacent to the right hilum in the medial segment of the middle lobe. **Fig. 2.**—Case 1. A lateral view showing a bronchogenic cyst between the major and minor fissures.

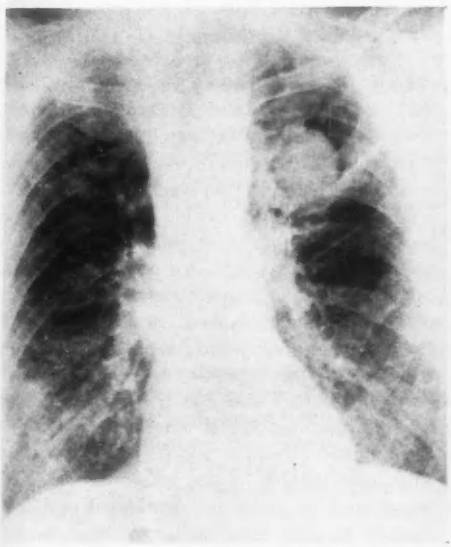


Fig. 3

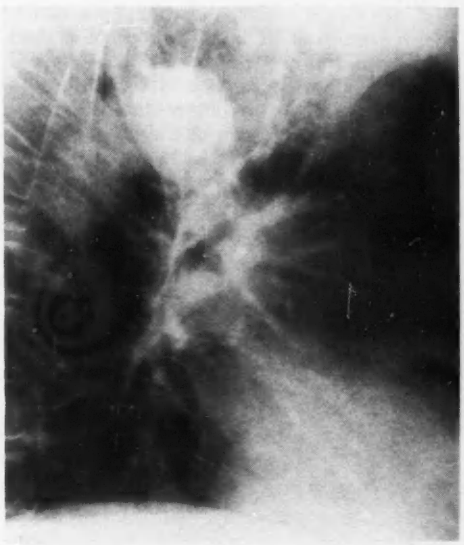


Fig. 4

Fig. 3.—Case 2. An aspergilloma in a bronchogenic cyst of the apicoposterior segment of left upper lobe. There is a conspicuous absence of hilar lymphadenopathy. **Fig. 4.**—Case 2. A lateral view of the bronchogenic cyst lying at the level of the aortic arch.

muscle and fine plates of bronchial cartilage, typical of bronchogenic cyst.

CASE 3.—The patient, a full-term female infant weighing 6 lb., remained pallid and limp after normal delivery. Breath sounds were not heard; the child failed to respond to resuscitation, and died one hour later. At autopsy it was found that congenital absence of the left half of the diaphragm had allowed herniation of most of the small intestine, transverse colon, spleen, body and tail of pancreas and left lobe of liver into the thorax. A tense, rounded semi-translucent cyst, 1 cm. in diameter, was found lying immediately anterior to the aorta and just above its passage through the diaphragm. No pedicle of attachment to the esophagus or lung was detected and the mass was partly covered by left pleura. Its wall was paper-thin with a white smooth inner lining and contained clear mucus. Microscopically the appearances were similar to those in Case 2. The cyst was easily movable and the respiratory tree and esophagus were intact. No muscle was present in the wall.

Esophageal Sequestration Cysts

CASE 4.—The patient, a 43-year-old man, complained of a "persistent cold in the chest", but apart from an irritating dry cough he felt well. He sought roentgenographic examination of his own volition because his mother had died from bronchogenic carcinoma. Physical examination was negative and there were no signs of weight loss or finger-clubbing. However, the roentgenogram of the chest showed an ovoid, homogeneous, soft tissue density, about 3.5 cm. in diameter, which was apparently in the posterior basal segment of the left lower lobe of the lung. No atelectasis was detected and at fluoroscopy the diaphragm was not fixed. An early bronchogenic carcinoma was suspected and exploratory thoracotomy was carried out. A tense cyst lay behind the heart immediately adjacent to the left wall of the lower esophagus and it was partly covered by left parietal pleura. These structures were dissected away with difficulty, and nutrient blood vessels from the posterior mediastinum were ligated. The entire muscle wall of the esophagus did not continue over the cyst, which had forced the left lower lobe of lung forward. No mucosal, esophageal or bronchial communications were demonstrated and the unruptured cyst was excised with a good deal of difficulty (Fig. 5). The wall was 0.1 cm. in thickness with a smooth pale inner lining and opaque mucous content. Microscopically, a single layer of pseudostratified columnar ciliated epithelium merged and alternated with a clear-cell squamous epithelium, each of which covered about half of the internal circumference. Clear nonciliated squamous epithelium was also seen in the attached glands, with occasional simple mucous glands lying between, and occasionally passing completely outside two distinct coats of plain muscle in the wall. On serial section, a few thin plates of bronchial-like cartilage were also detected in this muscle. These are the features of cysts of fetal esophageal sequestration type. The patient has remained symptom-free during two years of observation.

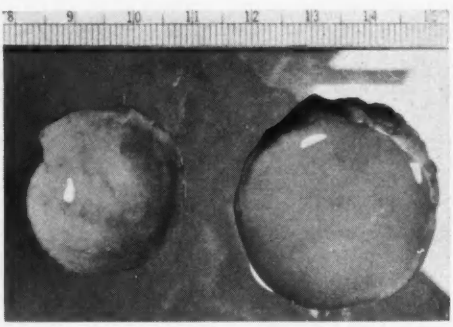


Fig. 5.—Case 4. The resected esophageal sequestration cyst showing the muscular wall and mucous content in the right half of the cyst.

CASE 5.—The patient, a seven-month-old male infant, had "rattling respirations" since birth unrelated to activity. The pregnancy, delivery and subsequent development of the child were normal. Two weeks before hospital admission, the child became so breathless that he needed frequent rest periods during each feeding. A paroxysmal dry cough developed, but it ceased after penicillin therapy. Physical examination showed no abnormalities. Roentgenographic examination demonstrated widening of the superior mediastinum to the right (Figs. 6, 7 and 8). A mass lay behind the trachea, pushing it anteriorly just below the thoracic inlet and compressing its lumen. The right margin of the mass was rounded, sharp and of uniform density. Esophageal fluoroscopy and film studies showed marked deviation of the esophagus to the right and slightly posteriorly. The maximum separation from the trachea in the lateral projection was approximately 3 cm., and the esophageal

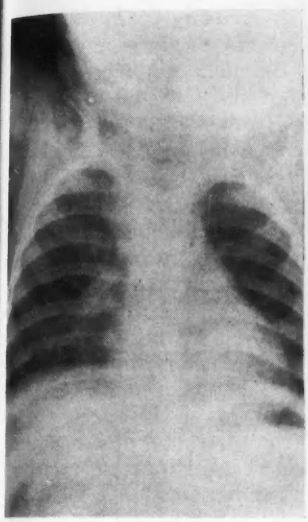


Fig. 6



Fig. 7

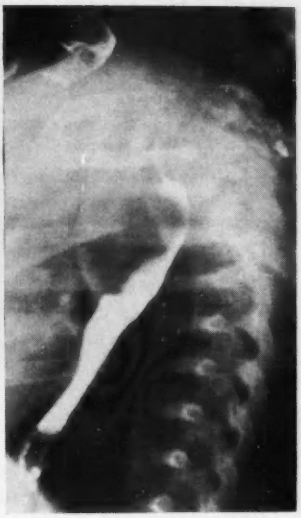


Fig. 8

Fig. 6.—Case 5. The widening of the superior mediastinum to the right is evident. **Fig. 7.**—A spot film taken during fluoroscopy showing marked right-sided deviation of esophagus in the superior mediastinum, which returns to the normal position at the level of the carina. **Fig. 8.**—Lateral film taken during fluoroscopy. A cyst lies between esophagus and trachea, bulging into esophageal lumen and displacing it posteriorly. The trachea is arched anteriorly and its lumen is narrowed.

lumen was also compressed by part of this mass which measured approximately 4 x 3 cm. The following possibilities were suggested; congenital tracheal or esophageal cyst, teratoma or tumour of the thymus.

An exploratory thoracotomy revealed a soft cyst densely adherent to the esophagus. No communication with the displaced trachea was seen. The phrenic and vagus nerves with their adjacent pleura were dissected medially and anteriorly until complete separation from the trachea was obtained. The cyst, 5 x 4 cm. in diameter, was intramural in the esophagus over a 3 cm. segment. Therefore, a strip of pleura was left on the esophagus and the posterior aspect of the cyst was dissected free so that it could be removed completely. An extensive defect in the muscle wall of the esophagus in its right anterior and mediastinal aspect which had previously been covered by the cyst was now demonstrated. The esophageal epithelium was intact and no further muscle approximation was carried out. The pleura overlying the esophagus was sutured and the azygos vein left undisturbed. Microscopically this intramural and extramucosal esophageal inclusion cyst showed features which were similar to Case 4. On further search no voluntary muscle was detected in its wall. The convalescence and recovery of this patient were uneventful.

Discussion

Because the final diagnosis in radiologically demonstrated thoracic coin lesions often depends on exploration,[4] the attachments of such cysts, which depend upon their embryologic derivation are important. The most common is the lung-bud or bronchogenic cyst. During fetal life, lung-buds are the first to protrude from the ventral aspects of tracheal groove of the fetal pharynx *before* the trachea finally separates. Isolated inclusions in these buds are often carried out into the lungs where they may form cysts which characteristically move with respiration on fluoroscopy.[5] They are thin-walled and round; are not usually in direct communication with the bronchi; are filled with mucus, and tend to occur near the primary and secondary bifurcations of the bronchial trees. While these cysts are usually isolated from the bronchi, minute bronchial communications do occur occasionally; secondary infection is rare but has been reported by Herrmann, Jewett and Galletti.[6] Secondary aspergilloma or "fungus-ball" formation seems to have a predilection for these cysts,[7] as in

our Case 2. Lobulation, due to rings of cartilage in the cyst walls, is very uncommon, although thin plates of cartilage are often seen microscopically with a small amount of plain muscle and occasional bronchial glands. One feature of note is the failure of the cysts to cause peripheral atelectasis, unlike bronchogenic carcinoma. The attachments to surrounding lung tissue are relatively slight and surgical removal is uncomplicated as a rule.

This variety of cyst commonly causes no symptoms until adult life when a paroxysmal cough may be provoked if an increase in the fluid contained in the cyst is sufficient to cause traction during changes in posture. The cysts never contain heterotopic gastric mucosa and thus do not result in hemoptysis from peptic ulceration. Some of the synonyms recorded are tracheobronchogenous, paratracheal,[8] paraesophageal and hilar cysts.[9]

Esophageal sequestration cysts probably arise at an earlier stage,[10] and are often confused with the bronchogenic type on the basis of certain shared histological features. It is realized seldom that the primitive fetal esophagus is itself lined by ciliated columnar epithelium for some time[11] and this has persisted even into adult life on rare occasions.[12] Sequestrations from it produce the intramural and paraesophageal cysts of mediastinum; these have been interpreted in various ways. Characteristically, an epithelial lining of respiratory-like ciliated columnar cells alternates with a stratified squamous epithelium made up of esophageal-like clear cells. Fine plates of bronchial cartilage occur in the walls, but with single or double coats of plain and even some voluntary muscle. The peculiar persistence of the multipotential qualities of the fetal gullet in such cysts suggests a neoplasm-like insensitivity to the influence of chemical organizers. Malignant transformation in these cysts has never been substantiated so far as we are aware as opposed to teratomata. However, it is a good illustration of the common origin of the bronchial tree and adult esophagus from primitive fetal gullet.

In some instances no columnar ciliated epithelial component is present and such cysts cause no difficulty in identification. It will be seen that the mere presence of *some* ciliated columnar epithelium in a lining or bronchus-like cartilage in the wall is not sufficient to establish bronchogenic origin. *All* components must be considered.

The muscle walls of these cysts are often continuous with that of the esophagus, so characteristically on radiological examination they move with swallowing rather than with respiration.[13] Being paraesophageal they may obstruct breathing or swallowing in infancy, and thus by virtue of their position produce symptoms much earlier than the bronchogenic cysts. Surgically their removal can be expected to be difficult and convalescence prolonged, for there is almost certain to be an extensive deficit in the esophageal muscle wall. The cysts contain mucus but never gastric juice, so that rupture during removal is not dangerous. As might be expected, they are associated rarely with true bronchogenic cysts or tracheoesophageal fistula. When this occurs, they are variously recorded as tracheoesophageal,[8] or "mixed" cysts,[14] and even as "teratomata" when they have a conspicuous mixture of respiratory and esophageal features.

A third variety of thoracic cyst, which is fortunately rare, arises from fetal remnants. It has been the source of further confusion, yet it should not, because radiologically it is characteristically associated with *cervical* vertebral anomalies such as anterior spina bifida. This type of cyst appears to arise in the early fetal cervical region because of an adhesion of cells of the primitive esophagus to the notochord, which arches forward and reaches the anterior surfaces of cervical vertebral bodies at this level.[15] As the thoracic viscera enlarge, the fetal cervical-esophageal tract elongates and traction cysts may descend for many segments, even into the abdomen. The cyst is often incorporated within the muscle coat of the esophagus, but it may be separated almost entirely when it is limited usually to the posterior esophageal region. A characteristic of this cyst is the presence of twin muscle coats and even Auerbach and Meissner's nerve plexuses. A fibrous band sometimes exists between the apex of

an elongated cyst and its site of origin in an anterior cervical spina bifida. In an analogous way the origin of the diaphragmatic muscle is marked in adults only by cervical phrenic nerve supply. These cysts are seldom asymptomatic, for their lining is capable of wider differentiation—into small bowel epithelium or more often heterotopic gastric mucosa. If the gastric lining predominates, its presence may lead to peptic ulceration with erosion through the cyst wall into surrounding lung tissue with hemoptysis resulting.[16] It is very important to have preoperative diagnostic radiological evidence of these, for rupture with leakage of gastric juice during operation can be disastrous. Until their embryologic origin was traced to the upper cervical region by Fallon, Gordon and Lendrum, these so-called "gastrogenous" cysts had no satisfactory embryological explanation. They were often classified, because of their position, with esophageal sequestration cysts, but the latter have no association with cervical vertebral anomalies and contain no heterotopic gastric mucosa.

Because of their obscure nature, this variety of cyst has been described by a confusing array of synonyms, for example –"duplications" and enterogenous cysts,[17] enteric cysts,[4] archenteric cysts,[19] and mediastinal enterocystoma.[18] Clearly much more attention should be paid to the site of the cyst and its covering layers, and complete reliance should not be placed on the histological appearance of the cyst lining. Of all the literature at our disposal we found that the reviews of Abell[8] and Fallon, Gordon and Lendrum[15] were the clearest and most succinct in their definitions.

Summary

Thoracic cysts of fetal origins have been described under a confusing plethora of names. A separation of these cysts has been attempted on a basis of location and by the predominating histological characteristics of their epithelial linings. As the cysts arise often from cells which are multipotential, their linings may include respiratory, esophageal, gastric, or small bowel mucosa. The classifications mentioned above are without surgical value. Three cases of bronchogenic lung-bud and two esophageal sequestration cysts have been reported in order to compare and contrast their diagnostic and surgical characteristics, which depend upon their embryologic origins. One of the former contained an aspergilloma or "fungus ball". The characteristics which allow differentiation of the so-called gastrogenous cysts of the notochordal traction type are also described.

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RÉSUMÉ

Les kystes endothoraciques d'origine embryonnaire ont été décrits sous une foule de dénominations diverses qui sont passablement confuses. Une classification des ces kystes a été essayée en se basant sur leur localisation et sur les caractéristiques histologiques les plus importantes de leur revêtement. Comme il s'agit souvent ici de cellules qui ont de nombreuses possibilités de différentiation, ce revêtement peut comporter des cellues de types respiratoire, œsophagien ou muqueux de l'intestin grêle. Ces classifications n'ont cependant qu'une faible valeur au point de vue chirurgical.

Trois histoires de cas de kystes bronchogéniques à partir d'un bourgeon pulmonaire et deux de kystes d'origine œsophagienne sont décrits ici pour comparer leurs diagnostics et leurs caractéristiques chirurgicales, qui dépendent de leur origine embryonnaire. Dans l'un de ces cas il fut trouvé une tumeur fungique, un aspergillome.

Les caractères différentiels qui permettent de distinguer les kystes dits gastrogènes et ceux par traction à partir des résidus notochordaux sont exposés en détail.

NITROGEN MUSTARD IN TREATMENT OF METASTATIC CARCINOMA OF THE TESTIS*

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CHEMOTHERAPY has assumed an important role in the control of malignant disease in recent years. Although these drugs may produce a prolonged remission in selected cases of cancer, chemotherapy alone has not cured any neoplastic process to date.

Historically, potassium arsenate (Fowler's solution) was used to treat chronic myelogenous leukemia almost 100 years ago. The era of modern-day chemotherapy began 20 years ago when Huggins and coworkers demonstrated the value of estrogen therapy in the management of metastatic carcinoma of the prostate. A vast amount of research is now directed towards cancer chemotherapy in general.

There are four main groups of anticancer drugs (Table I).

TABLE I.—CANCER CHEMOTHERAPY

1. Cytotoxic alkylating agents (nitrogen mustard)
2. Antimetabolites
3. Hormones
4. Antibiotics

1. *The cytotoxic alkylating agents.*—These have an available alkyl group which is incorporated into some crucial part of the cell nucleus and produces chromosomal fragmentation and faulty reunion. They act as mitotic poisons which interfere with cell division and are most toxic to cells in the proliferating phase. Because their mode of action is similar to that of radiant energy, they are also known as radiomimetic drugs. Included in the group are the mustard gas analogues, of which nitrogen mustard is the well-known prototype. These drugs are used predominantly in the treatment of the lymphomas and chronic leukemias. Nitrogen mustard is also of value in the control of malignant effusions when injected into the pleural or peritoneal cavity.

2. *Antimetabolites.*—Nucleoproteins are present in large amounts in neoplastic tissue. The antimetabolites act by substituting themselves for some essential component of the cellular nucleoprotein, producing the cell's destruction. For example, folic acid is necessary in the production of nucleic acid and when a modified form of this vitamin such as aminopterin is given, the normal utilization of folic acid is prevented, leading to cell necrosis. The antimetabolites are used especially in the treatment of acute leukemias. A member of this group, amethopterin (Methotrexate), is used in the treatment of choriocarcinoma.

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Presented by Dr. Ankenman at the Annual Meeting of the Canadian Urological Association, July 3, 1960, Banff, Alta.

3. *Hormones.*—These create an endocrine environment unsuitable for certain neoplasms. The palliative therapy of metastatic carcinoma of the prostate with estrogens is the most widely used form of hormonal cancer chemotherapy and needs no elaboration here. Androgens in premenopausal and estrogens in postmenopausal women with advanced breast carcinoma are used widely. The indications for use of adrenal steroids and their mechanism of action in the treatment of cancer is not so clearly defined.

4. *Antibiotics.*—Actinomycin D has recently been reputed to be of some value in the treatment of Wilms' tumour.[1] Other antibiotics are being evaluated in an intensive search for new and more effective agents.

Chemotherapy of Testicular Tumours

A survey of urological literature reveals few encouraging reports on the use of chemotherapy in the treatment of testicular cancer.[2]

1. *Nitrofurazone.*—This has been reputed to be of value in eight reported cases.[3] From the experimental work it would appear that this drug will not assume major importance in the treatment of testicular tumours.[4]

2. *Amethopterin* (Methotrexate).—This has prolonged remissions in women with metastatic trophoblastic tumours and possibly some patients may have been cured. Unfortunately, the testicular trophoblastic tumours have not shown a similar response to this drug. Six cases of choriocarcinoma of the testis, in which the drug has been used, have been reported.[5] All the patients have died and in only one was the titre of gonadotropin reduced.

At the Vancouver General Hospital, a patient with pulmonary metastases from a choriocarcinoma of the testis was given a total of 650 mg. of amethopterin. The Friedman test for the presence of gonadotropin reverted from positive to negative. The pulmonary lesions temporarily regressed but the pregnancy test again became positive and he went on to die. Because of the inherent dangers of bone marrow depression with this drug, marrow was obtained before treatment and stored for transplant if necessary, but it was not required.

3. *Combined Drug Therapy.*—Treatment of metastatic cancer of the testis by combined drug therapy has been reported recently, using different combinations of an alkylating agent, an antimetabolite and actinomycin D.[6] Twelve out of 23 patients so treated gave evidence of tumour regression.

TABLE II.—Nitrogen Mustard (HN_2)

Dosage:	0.2 mg./kg. intravenously, repeated in 24 hrs.
Toxicity:	1. Nausea and vomiting within hours. 2. Leukopenia, possibly anemia and thrombocytopenia within 3 weeks.

4. *Nitrogen Mustard.*—We wish to report our limited experience with nitrogen mustard (Table II). This drug is given intravenously in a dosage of 0.2 mg./kg. and is repeated in 24 hours. Care should be used in its administration because extravasation at the site of infusion may produce local sloughing. Within a variable period up to several hours after the injection the patients become nauseated and 50% of them vomit. Within one to three weeks a moderate leukopenia occurs and sometimes anemia and thrombocytopenia develop which require repeated transfusions.

Case 1.—The history of this 29-year-old patient was particularly interesting because the left testis had never descended and was not palpable. Also, there was a history of a right-sided mumps orchitis in 1945. In the spring of 1955 the patient noted enlargement of the atrophic right testis and at the same time complained of increasing weakness and fatigue, decreasing libido, and finally impotence. There was also progressive loss of facial and body hair so that he shaved only once every four or five days, whereas previously he had shaved daily. By the autumn of 1955 he felt a lump in the left supraclavicular area, but medical advice was not sought until April 1956, one year after the onset of symptoms. When first examined, a firm mass, 15 cm. in circumference, was discovered which replaced the right testis. A mass, approximately 10 x 7 cm., was palpated in the right mid abdomen, and bean-sized left supraclavicular lymph nodes were also palpated. A radiograph of the

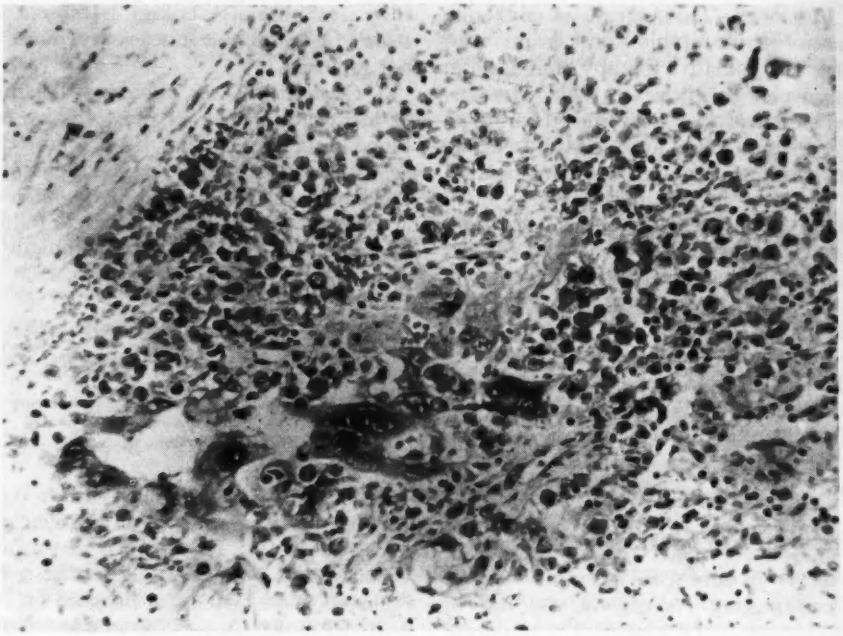


Fig. 1.—A section of the testis showing replacement by seminoma, which contains foci of choriocarcinoma.

chest and the Friedman test were negative. An intravenous pyelogram demonstrated the mass in the right abdomen. When orchiectomy was performed, the testis was found to be replaced by a tumour, which on pathological examination was a seminoma with foci of choriocarcinoma (Fig. 1). A biopsy of one of the supraclavicular lymph nodes was done and reported as completely replaced by seminoma. Radiation therapy, using the cobalt unit, was directed to the supraclavicular area and abdomen. The mediastinum was not treated. The palpable supraclavicular lymph nodes regressed, but the abdominal mass was still readily palpable and evident on radiological examination three and one-half months after radiation therapy. Nitrogen mustard, 30 mg., was administered intravenously in divided doses on two successive days and within three months the mass was no longer palpable or discernible on roentgenography. This patient has been followed-up carefully. He is alive and well four and one-half years after treatment, without radiological or clinical evidence of residual disease. On testosterone cyclopentylpropionate (Depotestosterone) 300 mg. every three weeks, his libido has returned to normal and he shaves daily.

Case 2.—A 27-year-old man became aware of an enlargement of the left testis in June 1958. The chest radiographs, intravenous pyelogram and Friedman test were negative. An orchiectomy was performed and the pathological diagnosis was embryonal carcinoma of the testis (Fig. 2). There was no clinical evidence of metastatic involvement and a radical retroperitoneal lymph node dissection was planned if feasible. However, at exploration, inoperable lymph node metastases were found around the left renal pedicle. A biopsy of these nodes showed metastatic embryonal carcinoma and a course of radiation therapy was directed to the para-aortic area. At the completion of this treatment a small lymph node was palpable in the left supraclavicular area and further radiation therapy was given to include this site and the mediastinum. He remained clinically well for six months but a routine chest radiograph in January 1959 showed widespread pulmonary metastases (Fig. 3). Early in February 1959, 26 mg. of nitrogen mustard was administered intravenously in divided doses on two successive days and the metastases regressed rapidly. The radiograph of the chest taken one year after treatment was negative (Fig. 4). This patient

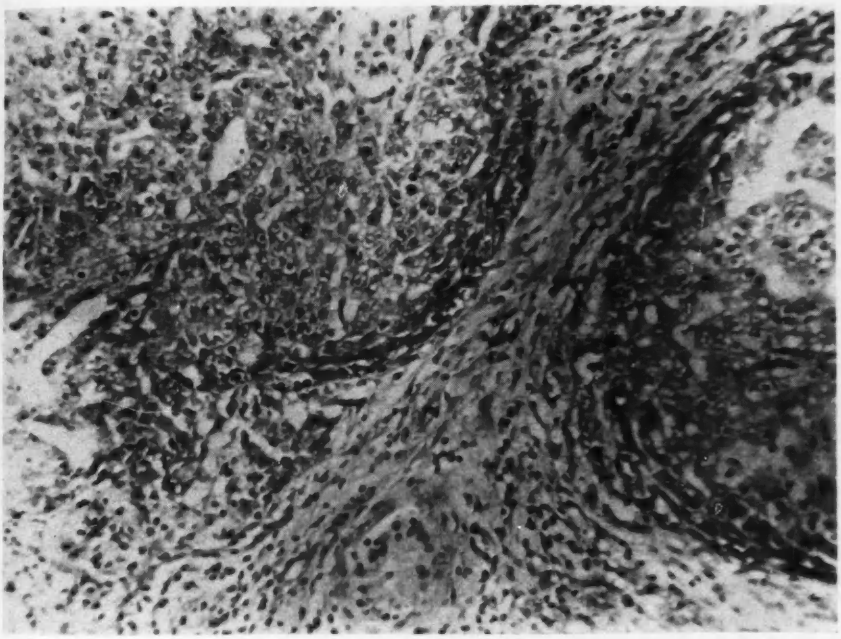


Fig. 2.—A section showing embryonal carcinoma of the testis.

is alive and well and gainfully employed two years after regression of metastases. However, a recent chest radiograph shows a solitary recurrent lung lesion.

Case 3.—A 42-year-old man noticed a mass in the left testis in April 1959. A radiograph of the chest, an intravenous pyelogram and the Friedman test were negative. An orchiectomy

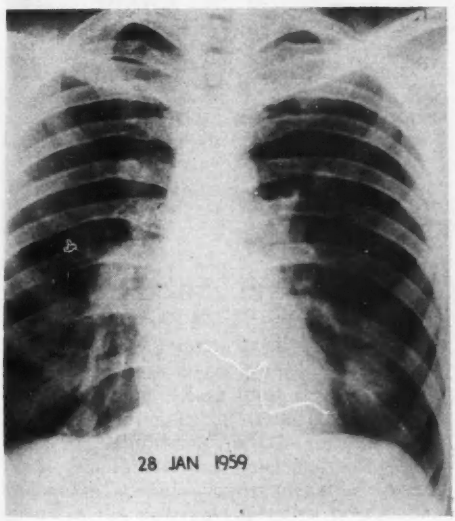


Fig. 3

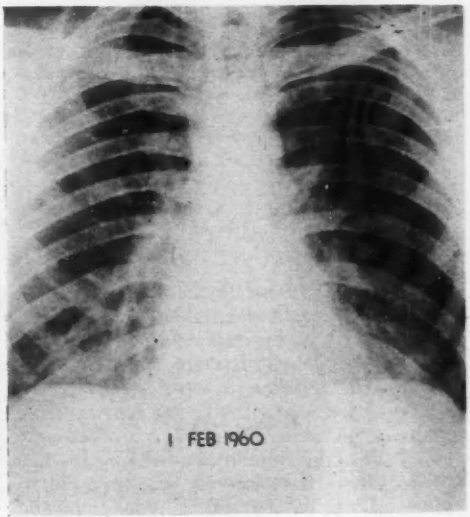


Fig. 4

Fig. 3.—A radiograph of the chest in January 1959 showing widespread pulmonary metastasis.
Fig. 4.—A radiograph taken in February 1959. Note the response of the metastasis to treatment.

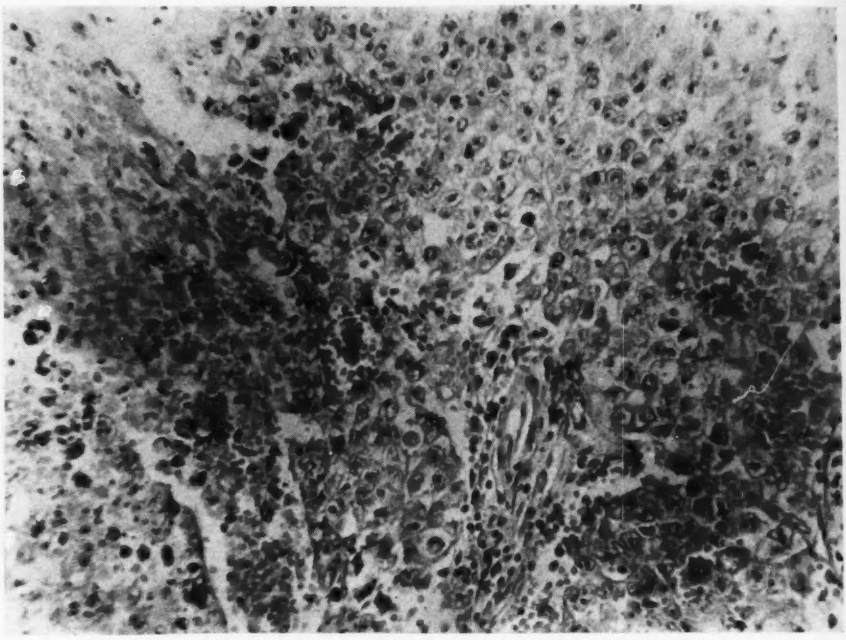


Fig. 5.—A section showing embryonal carcinoma of the testis.

was performed, and the pathological diagnosis was pure embryonal carcinoma (Fig. 5). The retroperitoneal space was explored, but inoperable para-aortic lymph nodes were found. A course of cobalt radiation therapy was given to the abdomen and completed by the end of June 1959. He remained well until December of this year when a small lymph node was

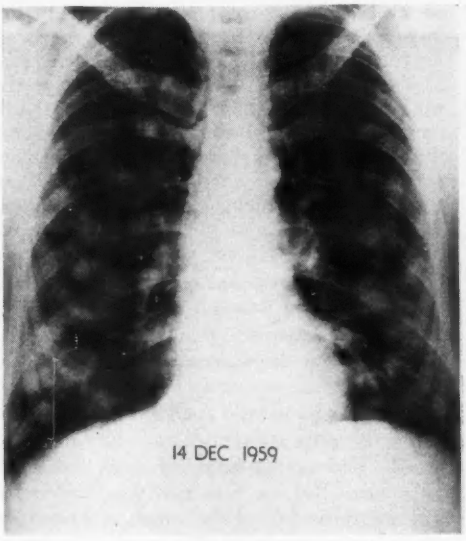


Fig. 6

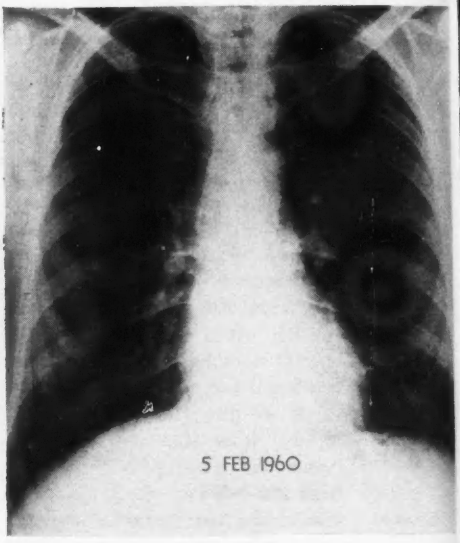


Fig. 7

Fig. 6.—A radiograph of the chest in December 1959 which shows extensive pulmonary metastasis. **Fig. 7.**—A repeat radiograph four weeks after treatment demonstrates the almost complete clearing of the lesions.

palpated in the left supraclavicular area. A chest radiograph at this time showed metastatic pulmonary disease (Fig. 6). Nitrogen mustard was given intravenously over a four-day period in divided doses in the total amount of 28 mg. Within four weeks the lymph node in the neck was no longer palpable and there was almost complete regression of the pulmonary metastases within six weeks (Fig. 7). The patient was given additional radiation to the lung fields. He did not improve following the combined attack on his disease, and because of bone marrow depression repeated transfusions were required. The patient returned to his home in northern B.C. and died very suddenly six months after treatment. Because of incomplete follow-up study, it is uncertain whether death was due to the effects of malignant disease or to bone marrow failure following therapy.

SUMMARY

Three cases of metastatic testicular carcinoma have shown response to nitrogen mustard therapy. Although the results are encouraging, no valid conclusions can be drawn from so few cases. Because these are uncommon forms of malignancy, it is difficult for one centre to accumulate sufficient cases to enable them to present results that have statistical significance. The paper is presented to stimulate interest in the use of chemotherapy in similar cases and to encourage the reporting of results.

The authors would like to acknowledge that the chemotherapy reported here was carried out under the auspices of the British Columbia Cancer Institute.

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RÉSUMÉ

Dans cet article, trois cas de cancer du testicule traités par la moutarde azotée sont présentés. Il s'agit d'un homme de 29 ans porteur d'un séminome du testicule droit avec métastases lointaines (creux axillaire) traité par la moutarde azotée et la radiothérapie au cobalt. Le deuxième cas est celui d'un homme de 27 ans présentant des symptômes semblables du testicule gauche. Enfin un homme de 42 ans porteur d'un carcinome embryonnaire également du testicule gauche. Ces trois cas ne peuvent pas être considérés comme des succès, mais ils furent améliorés et les résultats sont considérés comme encourageants.

Il est difficile de tirer des conclusions de séries aussi petites; la rareté de ce genre de tumeur rend d'ailleurs difficile à un seul centre, d'accumuler des données réellement valables. Cet article a surtout été écrit pour stimuler l'intérêt du corps médical dans l'emploi de ces nouvelles drogues.

SPONTANEOUS INTESTINAL PERFORATION IN THE NEWBORN*

"Intestinal perforation is not uncommon in the newborn baby and may occur before birth. When it occurs before birth it gives rise to meconium peritonitis, a sterile chemical process which is different from the bacterial peritonitis that follows post-natal perforation . . . Perforation is readily understandable when it occurs in the distended intestine proximal to an acute obstruction. In the absence of obstruction it is difficult to understand, is less frequently encountered and scant reference has been made to it in textbooks of pediatric surgery or in the medical literature of the last ten years. Its rarity is probably more apparent than real, for all cases may not have been reported . . . With advances in anesthetic techniques, in chemotherapy and in the understanding of the physiological needs of the newborn baby, perforation is now amenable to surgical treatment with a fair prospect of success. Awareness of it will lead to earlier diagnosis, the factor of greatest importance in securing a successful outcome."

*GANDHI, R. K.: *J. Roy. Coll. Surgeons Edinburgh*, **6**: 293, 1961.

EPITHELIOMA COLLOIDE DU SEIN

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On ne peut expliquer les caprices de l'évolution du cancer du sein, et les méthodes de traitement se modifient continuellement, tant du côté de la chirurgie que de la radiothérapie, l'hormonothérapie et la chimiothérapie. L'optimisme dans le pronostic du cancer du sein est souvent transitoire et on demeure perplexe devant la diversité et la non-concordance des résultats obtenus.

Bien que l'étude du pronostic du cancer du sein, en regard du type histologique, ait déjà été faite,[1, 2] il nous a cependant paru intéressant de présenter un groupe de cancers mammaires, les épithéliomas colloïdes.

TABLEAU I.

	Age	*Sexe*	*Durée*	*Macroscopie*	*Ganglions*	*Intervention*	*Evolution*
1	40	F	12 ans		envahis	mastectomie radicale	décédée
2	43	F	11 ans		négatifs	radicale	décédée
3	48	M	9 ans	5 cm.		simple	vivant (métastases osseuses)
4	77	F	6 ans	1 - 2 cm.	négatifs	radicale	vivante
5	54	F	9 ans	2.5 cm.	négatifs	radicale	vivante
6	40	F	7 ans	microscopique	négatifs	radicale	vivante
7	66	F	7 ans			simple	vivante
8	79	F	4 ans	3.5 x 3 cm.	négatifs	radicale	vivante
9	75	F	?			simple	?

Définition

Le terme colloïde se rattache à l'aspect macroscopique d'un néoplasme et provient de la coloration brun pâle et de la transparence du tissu tumoral, ressemblant à la colloïde thyroïdienne. Aussi, pour certains, l'épothélioma colloïde est une entité purement macroscopique.[3] C'est une tumeur à contours généralement bien définis, parfois lobulée, d'aspect gélatineux; au toucher, la surface de coupe est visqueuse. Pour d'autres, l'épithélioma colloïde de la glande mammaire correspond à une entité microscopique particulière,[4, 5, 6] dont la caractéristique prédominante est l'abondante sécrétion de mucus par les cellules tumorales. Ce mucus est parfois intracellulaire comme dans les épithéliomas mucipares du tube digestif (cellules en châton de bague); ou bien il est dans les lumières glandulaires, ou encore dans le tissu péricanaliculaire ou périglandulaire; dans ce dernier cas, le mucus est très abondant et forme des plages à travers lesquelles sont disséminées les structures épithéliomateuses glandulaires (Figs. 1 et 2). C'est à cette dernière forme histologique que la majorité des histopathologistes donne le nom d'épithélioma colloïde de la glande mammaire. Ce terme est toutefois réservé aux formes pures et non aux épithéliomas anaplasiques qui présentent ici et là cette évolution mucoïde.

Matériel

Dans les 700 cancers mammaires traités à l'Hôtel-Dieu de Québec, au cours des quinze dernières années, nous avons retrouvé neuf cas d'épithélioma colloïde, dont un cas chez un homme (Tableau I).

Age

Cette forme d'épithélioma mammaire semble se développer à l'âge habituel de l'épithélioma du sein.

Durée

La grande caractéristique de ce type d'épithélioma mammaire est son évolution lente. Deux patientes sont décédées; l'une (Cas 1), douze ans après mastectomie radicale avec évidement de l'aisselle, où

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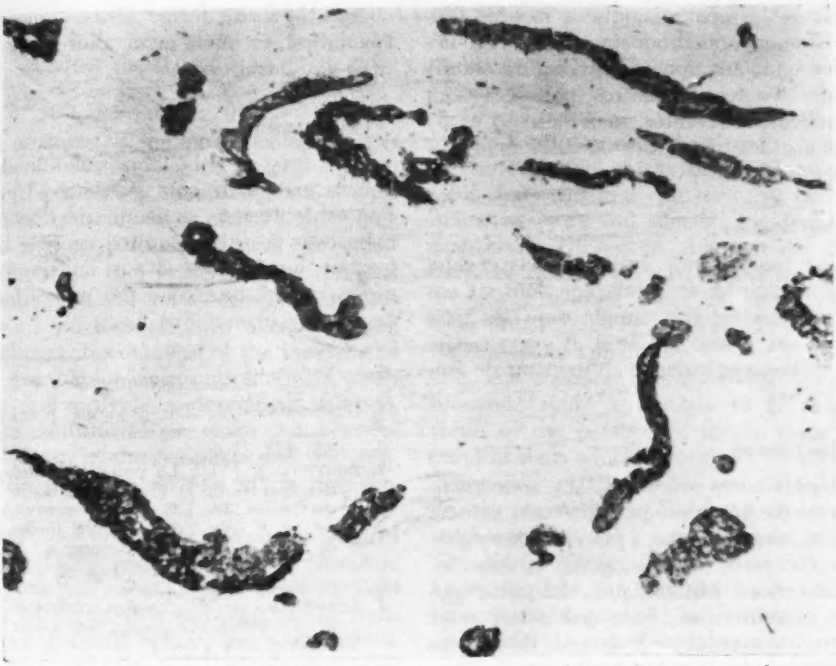


Fig. 1.—Cordons épithéliomateux baignant dans une abondante substance mucoïde.

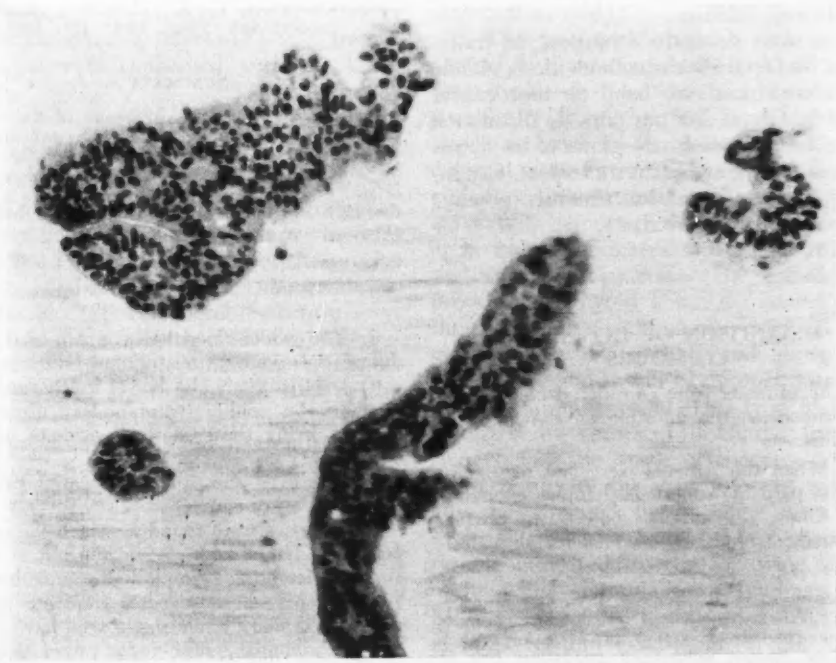


Fig. 2.—Cordons épithéliomateux baignant dans une abondante substance mucoïde.

l'examen histopathologique a montré l'envahissement ganglionnaire; l'autre est décédée onze ans après l'intervention chirurgicale. Toutes les autres patientes sont actuellement vivantes, mais l'une (Cas 3) présente des métastases osseuses multiples après neuf ans d'évolution.

Traitement

Une mastectomie radicale avec évidement axillaire a été pratiquée dans six cas et une mastectomie simple dans les trois autres cas. Dans le Cas 3, il y avait déjà des métastases osseuses au moment de l'intervention.

Discussion

L'épithélioma colloïde de la glande mammaire a été considéré par différents auteurs comme un néoplasme à pronostic favorable et a été placé dans le grade I, dans les classifications histologiques. La série que nous présentons ici, bien que petite, nous permet de conclure à la lenteur de l'évolution de ce néoplasme, mais il faut admettre sa malignité, le décès étant survenu dans deux cas et le troisième présentant des métastases osseuses.

Par suite de cette évolution, le traitement de l'épithélioma colloïde de la glande mammaire doit être celui de tout cancer du sein. On ne doit pas faire de distinction entre cette forme histologique et les autres formes dans le choix du traitement. Cependant, dans l'étude des résultats obtenus (évolution et morbidité), on doit tenir compte de cette forme histologique, dont l'évolution est nettement plus lente que celle des autres types.

Conclusion

Neuf cas d'épithélioma colloïde de la glande mammaire sont présentés. L'évolution est lente mais ce néoplasme donne des métastases à distance, aussi ce type histologique ne justifie-t-il pas un traitement moins radical que celui des autres formes de cancers mammaires.

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Summary

Nine cases of colloid carcinoma of the mammary gland are studied here. The evolution of these tumours is slowly progressive, but this form of neoplasia can metastasize far from the site of origin. For this reason this lesion requires a very thorough treatment, as complete as that used in other types of mammary carcinoma.

DEFINITION OF INTESTINAL TUMOURS*

"The tumours were considered clinically to be primary in the intestine on the following grounds:—

1. When the patients were first seen there was no palpable superficial lymphadenopathy.
2. Chest radiographs showed no obvious enlargement of the mediastinal lymph-nodes.
3. The white-blood cell-counts, total and differential, were within normal limits.
4. At laparotomy the bowel lesion predominated, the only lymph-nodes obviously affected being those in its immediate neighbourhood.
5. The liver and spleen appeared free of tumour in every case.

"Some cases were found in which the retroperitoneal lymph-nodes were affected as well as the mesenteric lymph-nodes and bowel. We excluded all these cases from our study since the primary lesion might well have been in the abdominal lymph-nodes rather than in the bowel."

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*Dawson, I. M. P., Cornes, J. S. and Morson, B. C.: Primary malignant lymphoid tumours of the intestinal tract: report of 37 cases with a study of factors influencing prognosis, *Brit. J. Surg.*, **49:** 80, 1961.

PREAURICULAR SINUS*

JOHN A. McLACHLIN, M.D., F.R.C.S.[C] and ROBERT O. FARLEY, M.D., F.R.C.S.[C], *St. Thomas, Ont.*

THE PROBLEM of the preauricular sinus is reviewed because it is the general surgeon who is most likely to be confronted with these cases and because of the general lack of awareness of this congenital abnormality. Probably this is due to the minimal disfigurement and the absence of symptoms arising from these lesions unless infection supervenes. Recognition of the presence of the preauricular sinus and adequate treatment will provide a permanent solution, whereas failure to recognize the underlying etiology of the symptoms will only perpetuate the problem and allow it to become worse.

There are two types of sinus in front of the ear. One, which the English literature refers to as "ear-pit", is a small straight sinus which is able to admit at the most the tip of a hair bristle; it is very shallow and does not require treatment. The other is the spiral-shaped preauricular sinus, which lies in close apposition to the cartilage either of the anterior part of the helix or the immediately inferiorly placed tragus. This paper is concerned with the latter entity.

EMBRYOLOGY

In 1825 Rathke[1] first described the embryology of the branchial apparatus in vertebrates. Over the years many theories have been advanced regarding the precise origin of branchial cleft abnormalities. There is still considerable controversy, which is not surprising since the differentiation of the whole branchial apparatus process takes place from the 10th to the 50th day of gestation. It first appears in the embryo of 2.6 mm. and has entirely disappeared by the time the embryo is 14 mm. in length.

This confusion regarding origin has led to the adoption of the term "lateral cervical sinus and cyst" for the very familiar branchial cysts and sinuses; the term lateral being chosen to differentiate them from the midline cervical anomalies. There are three distinct types of sinus which arise either from or in close proximity to the branchial apparatus.

1. The common lateral cervical sinus or branchial sinus is thought to arise as a result of the persistence of the transitory cervical sinus which is formed by a relative overgrowth of the second arch. This overgrowth starts during the sixth embryonic week and overlaps the more caudal arches. The sinus opening usually lies along the lower third of the anterior border of the sternomastoid muscle, but rarely it may open into the supratonsillar fossa forming a fistula. The tract passes back between the artery of the second arch (external carotid) and the artery of the third arch (internal carotid).

2. The rare type of sinus such as described by Byars and Anderson[2] and more recently by de Bord[3] is thought to come specifically from the first branchial cleft which accounts for its connection with the external auditory canal. The first branchial cleft forms the external auditory canal and its anterior boundary is the first branchial arch which bifurcates into the maxillary and mandibular processes. The second arch produces the hyoid bone. An anomaly of the first cleft should appear high in the neck below the mandible and above the hyoid bone and it will likely open into the external auditory canal. This type is characterized by recurrent discharge from a sinus below the angle of the jaw or the lower border of the mandible and is usually accompanied by drainage from the external ear. That this type of sinus is rare is indicated by the following series of branchial abnormalities; Hyndman and Light,[4] one in 90; Ladd and Gross,[5] one in 82; Neel and Pemberton[6] at the Mayo Clinic, none in 319 cases. Byars and Ander-

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*Presented at the combined Southwestern Surgical Association and the Regional Meeting of the Royal College of Physicians and Surgeons of Canada, London, Ont., November 1960.

From the Department of Surgery, University of Western Ontario, London.

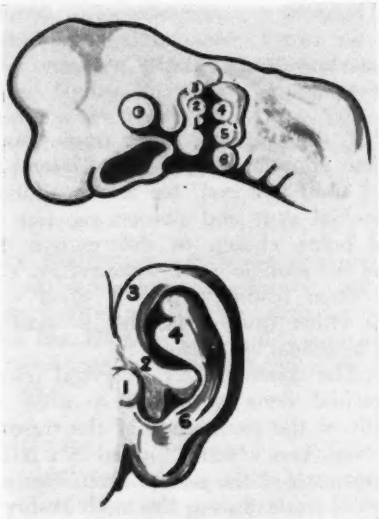


Fig. 1.—Development of external ear. *(Adapted from Figs. 508B and E in L. B. Arey's "Developmental Anatomy", 6th ed., W. B. Saunders Company, Philadelphia, 1954.)*

son reported three in 1951. Since then Anderson[7] has encountered two more.

3. The third type is the preauricular sinus. This has been loosely described as arising from the first branchial cleft, but specifically it arises in the formation of the ear from tissues immediately adjacent to the first branchial cleft.

Development of External Ear

The ear arises from the dorsal extremities of the first and second gill arches surrounding the first branchial cleft. The helix and tragus come from three circumscribed protuberances on the first gill arch while the antihelix and antitragus come from three similar formations arising in the second arch. Faulty fusion of these protuberances is generally considered to be the cause of the preauricular sinus[8] (Fig. 1).

Clinical Discussion

The most common clinical feature with which these cases present is acute inflammation. A history of previous excision is not uncommon. It has been stated that any type of cellulitis or abscess in the preauricular area should prompt a search for

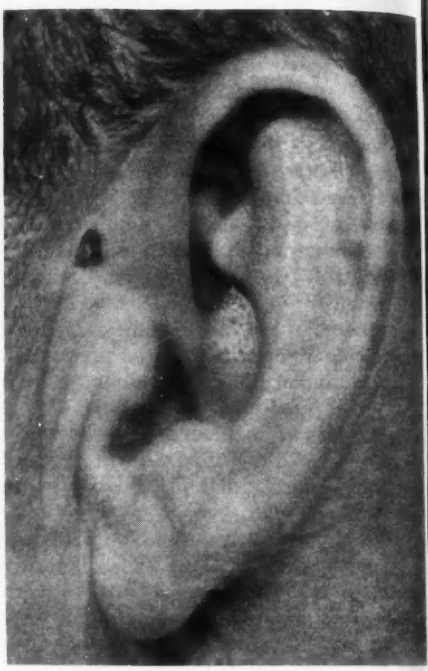


Fig. 2.—Preauricular sinus.

one of these sinuses, even though the infection may be at a distance. The basic cause may be a preauricular sinus quite close to the ear as in Fig. 2, where the orifice of the sinus has been delineated with ink for clearer demonstration. Recurrent drainage will persist until the whole sinus tract has been completely excised. An area of ulceration may develop in front of the ear in association with the blocking of one of these sinuses. Palliative treatment of the ulcer with fomentations, ointments or topical antibiotics will be ineffective. This paper is based on six patients with this problem. Two of these patents had bilateral lesions, making a total of eight sinuses.

There is a distinct hereditary element in this entity of preauricular sinus. It is inherited as an incomplete dominant of variable manifestation and without sex difference,[9] and in these six cases the two bilateral cases were in first cousins. One surgeon may see more than his share of these patients because the hereditary factor produces a relative excess in one locality.

TABLE I.

Cases	Age	Heredity	Bilateral	Repeated inflammation	Previous surgery
1. C.H.	2	Yes	Yes	Yes	No
2. J.H.	2	Yes	Yes	Yes	Yes
3. S.B.	19	No	No	Yes	Yes
4. R.B.	42	No	No	Yes	Yes
5. S.M.	6	No	No	Yes	No
6. N.C.	33	No	No	Yes	No

Treatment

The essential treatment of preauricular sinus is complete excision of the sinus tract. This is best done under general anesthesia, and in an adult endotracheal anesthesia is preferred. The region is quite vascular, and suction with a fine-tipped sucker will aid materially in providing a clear view of the tissues. We have preferred not to inject methylene blue, but to keep the plane of excision out in normal tissue. If the tract is filled with methylene blue and accidentally cut across, the surrounding tissue becomes stained and the disadvantages of this staining outweigh the value of defining the tract. The presence of the facial nerve is not a problem because the sinus generally lies above the tragus and the facial nerve runs anteriorly at a lower level. The cause of the repeated drainage is the congenital sinus and unless this sinus is completely removed recurrent drainage is almost inevitable. Some of the patients present themselves with an area of ulceration anterior to the orifice of the sinus. In one patient (Case 2) there was a raw area of granulation tissue to which her hair was so matted that it had to be cut free. It is not necessary to excise this area of ulceration but the sinus is attacked directly in the relatively healthy tissue in front of the ear. Once the sinus is removed, the ulcerated area will heal with surprisingly little scarring. If an unsightly scar remains subsequent plastic surgery performed on noninfected tissues should give a better cosmetic result. The sinus runs in close proximity to the cartilage of the anterior helix and usually appears to be adherent to it. To ensure that all the sinus is removed it is best to remove a shaving of cartilage with the sinus. A small rubber drain should be brought out through the lower end of the incision; this can be removed in from 48 to 72 hours without affecting the cosmetic result of the procedure. The first five patients treated in this series have been followed up from three to 11 years without recurrence. The lesion in the sixth patient healed by first intention, but has been operated upon too recently to include as a cure.

Discussion

The preauricular sinus is a congenital abnormality usually brought to light by the development of local infection. They are of interest to the general surgeon because he is the one most likely to see them. The exact origin of these sinuses is not clearly understood, but they certainly arise as components of or adjacent to the first branchial cleft. Recognition and complete excision of the sinus tract will lead to a cure.

Summary

The problem of preauricular sinus is presented along with a review of the embryological development. Six cases with a total of eight sinuses are reviewed. The hereditary factor is confirmed. The diagnosis should be considered whenever any preauricular infection is encountered. The necessity for complete excision of the tract is emphasized. If there is an area of infected ulceration present, this need not be attacked directly, but will heal with removal of the sinus tract alone.

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Résumé

L'existence du sinus préauriculaire et les troubles dont il peut être le siège devraient être connus des chirurgiens généraux, car ce sont eux qui ont le plus souvent l'occasion d'en rencontrer des cas.

Embryologiquement ce sinus dérive de la première fente branchiale ou plus exactement des tissus immédiatement adjacents à cette première fente, lors de la formation de l'oreille.

Ils se manifestent en général par des inflammations aiguës. Selon les auteurs, toutes les fois qu'une abcès ou qu'une poussée de cellulite apparaît dans la région préauriculaire, il y a lieu de soupçonner une anomalie de ce genre.

Six de ces cas sont présentés ici. Le traitement chirurgical est l'excision du trajet du sinus sous anesthésie générale avec intubation endotrachéale. Un drainage sera laissé en place. Les auteurs ont renoncé à l'ancienne technique qui consistait à injecter du bleu de méthylène dans le trajet, préférant exciser largement jusqu'au tissu sain devant l'oreille. Les cicatrices sont rarement gênantes, et si c'est le cas, il sera facile de corriger la situation par une intervention esthétique subséquente.

THE USE OF HYPOTHERMIA IN THE CLOSURE OF ATRIAL SEPTAL DEFECTS IN CHILDREN

The beneficial effects which could be expected from the use of hypothermia in cardiac operations were initially appreciated and demonstrated by W. G. Bigelow. The method was utilized by F. J. Lewis in the performance of the first successful "direct-vision" repair of an atrial septal defect in September 1952. At the present time, three different surgical methods are widely used to correct atrial septal defects—the atrial well, cardiac bypass, and general body hypothermia.

Mustard, Baird and Trusler (*Surgery*, 50: 301, 1961) reviewed their experience with the method of general hypothermia for the closure of atrial septal defects in children. At the end of June 1960, 157 patients with atrial septal defects had been operated upon at The Hospital for Sick Children, Toronto; hypothermia had been used in 105 of the operations. Atrial septal defects associated with total anomalous pulmonary venous drainage or with a separate ventricular septal defect are excluded from their review.

The results of the operation were as follows. The morbidity resulting from this procedure was low. There have been four cases of wound infection, of which only one, osteomyelitis of the lower sternum, was a cause for prolonged hospitalization. One patient developed a staphylococcal pericarditis which responded rapidly to treatment. No child in this series required postoperative bronchoscopic suction. Heart block did not occur, although one child had atrial flutter for three months.

There have been three cases of cerebral gas emboli, one with air and two with carbon dioxide. In two of the children the gas was also noted in the coronary arteries and ventricular fibrillation occurred during operation. The children remained unconscious for one to three days postoperatively. They were all treated by prolonged hypothermia, as described by Williams. All have recovered completely and have no detectable neurological sequalae.

Incomplete closure of the atrial septal defect was suspected clinically in two children and proved by postoperative cardiac catheterization. In one child, prolonged cardiac massage had been required to overcome ventricular fibrillation and it is believed that the recurrence occurred at this time. This recurrence was large enough to demand a second operative procedure. The other recurrence was small (10% rise in atrial oxygen saturation) and further treatment was not recommended.

In the entire series of 95 patients, there was only one death from the initial operation. This child died 16 hours after the operation (September 1957) from cardiac tamponade. The child with the severe recurrence described was operated upon a second time two years after the initial procedure; she died on the operating table from an atrial tear which occurred during the thoracotomy. Thus, the hospital mortality rate from this procedure was 2.1%.

The other 92 patients are thought to be entirely cured of the defect. Postoperative cardiac catheterization has been performed on 14 other patients and in none has there been evidence of a persistent left-to-right shunt.

PRIMARY BASILAR IMPRESSION OF THE SKULL*

H. F. W. PRIBRAM, M.A., M.B., D.M.R.D.† and
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CONGENITAL abnormalities at the foramen magnum have received increasing attention since Schüller[16] first described the anatomical features of basilar impression. Subsequent papers[4, 6, 12, 14, 18] have drawn attention to the fact that such abnormalities are not at all uncommon and that the diagnosis may be extremely difficult.

Primary basilar impression is a congenital abnormality. A genetic mode of determination has been suspected but only one account of two members of a family requiring surgery for this condition has been published.[17] Its appearance, therefore, in a mother and son who have both had posterior fossa decompressions is of interest.

Two further cases are reported as the clinical features are at times grossly misleading. In one case the early symptoms suggested myasthenia gravis.

CASE 1.—James K., a 19-year-old pastrycook, first attended the Central Middlesex Hospital on September 12, 1952. He had been in good health until three months previously when he began to stumble, catch his feet on uneven ground and lose his balance, particularly in the dark. His legs felt stiff and numb. He had no control over them; they would give way unexpectedly and he was unable to step up on to a chair. If he sat with his legs crossed he could not tell where his feet were and he would often get up without uncrossing them and fall over.

During the past year he had suffered from mild occipital headaches and latterly had noticed some weakness of grip. For one week he had had urgency of micturition, but he had no difficulty in swallowing and no change in speech.

On examination there was a well-marked nystagmus to right and left, wasting of both trapezii in their upper parts, and weakness of the sternomastoids particularly the left. The tongue showed marked fibrillation and the right side was wasted.

There was no wasting or weakness of upper limbs, but slight incoordination and clumsiness of fine finger movements. He had slight weakness and early clasp-knife spasticity of legs. There was no cutaneous sensory loss but severe loss of postural sense in toes and absent vibration sense up to and including the iliac crests. All tendon jerks in the upper and lower limbs were greatly increased and plantar responses were extensor. He walked unsteadily on a wide base, lifting his feet high, and Romberg's sign was positive.

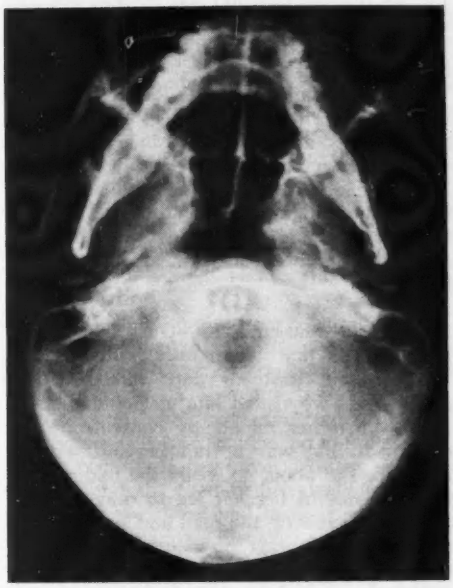


Fig. 1.—A basal view of skull (Case 1) to show the unusually small foramen magnum with encroachment along the anterolateral margin.

A clinical diagnosis was made of compression at the level of foramen magnum. Radiographs of the skull showed that the foramen magnum was extremely small with encroachment on both anterolateral margins suggestive of local vertebralization (Fig. 1). A diagnosis of basilar invagination was made; an operation was advised and was performed on October 14, 1952, by Mr. I. G. Roberts.

The patient was placed in the prone position with the head acutely flexed so that the chin rested against the sternum. There was a

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head tilt of 15° on the table. A midline incision from the external occipital protuberance to the spinous process of C.3 was made. The occipital bone, the posterior part of the foramen magnum and the arch of the atlas were removed to expose the dura over the cerebellar hemispheres. No gross abnormality or evidence of severe cord pressure was noted, so the wound was closed in layers.

Following operation there was no dramatic improvement but, on the other hand, no further deterioration. Before discharge from hospital, six weeks after operation, he said he felt a little steadier but there was no objective improvement in gait and no change in the physical findings. One year later he reported that his walking had improved a little; his feet still felt numb. He was free from headaches and his bladder function was normal. His gait was still ataxic on a wide base but was obviously better. Apart from this there was no change in physical signs.

He has continued under observation and when last seen in July 1959 had been working full time as a pastry-cook since 1954. His walking has improved considerably though he is still inclined to walk on a wide base. Apart from definite improvement in postural sense in the toes, his physical findings are unchanged and there has been no further progression of his disease.

CASE 2.—Margaret K.—Our first patient had told us that his mother suffered from "nerves", was unsteady and walked with a stick. When we visited him on September 30, 1952, she rather reluctantly consented to be examined. She was a frail nervous woman, aged 56. For about six years she had noticed the tendency of her left leg to drag, became nervous of crossing the road and began to use a stick. Three years previously she had developed difficulty in swallowing and nasal regurgitation which persisted for about one month. Since that time she had had occasional pins-and-needles parasthesiae in the hands and left leg. She also suffered from frequent pains at the back of the neck, which radiated into the head and were made worse by laughing.

In addition to James, she had seven children who were alive and well. Her mother, who had died at 49 years of age, had suffered from progressive unsteadiness, "trouble with her legs" and epileptic fits. One brother and one sister were alive and well and one brother had died of coronary thrombosis.

On examination the only abnormal signs were a marked increase in all deep reflexes and an extensor plantar response on the left. A radiograph of her skull was taken but, unfortunately, the films were unsatisfactory. She was asked to attend for further radiographic examination but did not do so and contact with her was lost until she was referred again by her own doctor on July 15, 1958, when her condition had deteriorated greatly.

Over the past five years she had developed an increasing tremor of the head. The difficulty in swallowing and nasal regurgitation had recurred. The unsteadiness in walking and weakness of her left leg had steadily increased, so that by December 1957, she could not walk without the help of one person and a stick. At Christmas 1957, she had an attack of coughing and since then had been much worse. Her left arm became weak and she could walk only if she was supported by two people.

On examination, she was a small frail woman who held her neck extended and had a constant coarse rotatory tremor of the head. The occiput was flattened. The pupils reacted normally, but the right was smaller than the left and she had slight right ptosis. There was well-sustained nystagmus to the right and a probable weakness of the palate. The left outstretched arm tended to fall away but there was no definite weakness. There was marked incoordination and intention tremor, greatest in the left. Both legs were spastic and weak, the left more than the right, with marked incoordination on purposive tests. All deep reflexes were greatly exaggerated and both plantar responses were extensor, but there was no sensory loss. Her gait was grossly ataxic and she could walk only with assistance.

Radiographs of the skull showed that the foramen magnum was small with encroachment anterolaterally. The petrous apices were elevated and the occipital condyles lay high in relation to the mastoid tips. The tip of the dens lay 11 mm. above Fischgold's line[7] and 11 mm. above McGregor's.[12] Bull's angle was 35°,[3] and the basal angle 130°. The posterior lip of the foramen magnum was infolded. There was very marked hyperextension of the cervical spine, but no associated abnormalities were noted.

Her brother later very kindly consented to attend the hospital for examination. He was completely asymptomatic but the findings on radiographic examination of the skull are of interest. The petrous apices were markedly elevated and the posterior lip of the foramen magnum was infolded. Measurements were as follows: the tip of the odontoid process and the atlanto-occipital joints lay 12 mm. and 10 mm. respectively above Fischgold's line.[7] It

was not possible to trace the remaining members of the family.

Operation was advised and performed on August 19, 1958, by Mr. T. G. I. James. In the sitting position, the foramen magnum was displayed. The atlas was tightly jammed against it and the axis was against the atlas in the same way. The occipital bone, the foramen magnum and the posterior arch of the atlas were nibbled away. A tight band of dura mater surrounding the dorsal aspect of the foramen magnum was divided over the right cerebellar hemisphere. There appeared to be no increase of pressure in the posterior fossa, herniation of the tonsils or abnormality of the fourth ventricle.

The postoperative improvement was steady and rapid for there was an immediate decrease in the head tremor and incoordination. One month after operation she was able to walk with a stick and had taken a few steps on her own. She had no difficulty in swallowing.

When last seen in July 1959, she was able to walk indoors without a stick, but liked to carry one when out of doors for reassurance. The cerebellar signs and head tremor had disappeared.

The clinical picture of compression at the foramen magnum is so varied that misdiagnosis is common. In the differential diagnosis myasthenia gravis is not previously recorded, and therefore the presentation of the following case is of interest.

CASE 3.—Pamela S., a 25-year-old woman, had been in good health until January 1958, when during a long walk in Lincolnshire, her chest felt tight and she became wheezy. These symptoms came on suddenly during the first episode and thereafter occurred at intervals of a month or so during the cold weather. The attacks came on with exertion when she hurried or ran and were regarded as mildly asthmatic.

In October 1958 she caught a cold; her nose was blocked and stuffy; her throat felt tight and dry, and her voice assumed a nasal quality. She consulted an ear, nose and throat specialist, who found a soggy nasal mucosa with bilateral nasal obstruction, and thought the findings were sufficient to account for her abnormal voice. With local treatment the nasal condition improved.

At Christmas 1958, her voice became much worse. It was now a strain to speak and there were times when she was unable to speak at all. She now had some difficulty in swallowing, particularly fluids, and these were often regurgitated through her nose. She tired easily; her arms were affected more than her legs. The patient had noticed that her voice was far better in the morning than at night, but that the more she talked the worse it became. She never had difficulty in swallowing at breakfast but found dinner a very difficult meal to eat. Her cough had been weak and feeble for the previous four to five months. She again consulted the otolaryngologist, who found a palatal and laryngeal palsy and referred her for a neurological opinion on January 28, 1959.

She was a pleasant intelligent girl and a good witness. Her posture was bad and the shoulders tended to droop and fall forward. The external ocular movements were present in the full range; there was no ptosis, diplopia or nystagmus. There was suspicious bilateral facial weakness with poor eye closure. The palate was immobile and gag reflex was weak, but there was no sensory loss. Her voice had a markedly nasal quality. She could count up to 100 but showed marked fatigue when she reached 60. The sternomastoids appeared thin, as did her arms, and on testing neck flexion against resistance, and power in the arms, she appeared to fatigue quickly. All deep reflexes were brisk but the plantar responses were flexor. Her chest expansion was poor, the breathing being almost entirely diaphragmatic.

She was admitted for investigation with a provisional diagnosis of myasthenia gravis. Tomograms of chest showed no evidence of thymic tumour. A test dose of neostigmine (Prostigmine) produced no significant improvement. It is true she seemed to fatigue less when counting, her eye closure seemed stronger and she thought her swallowing was easier, but she developed marked generalized muscular twitching. Electromyographic tests performed on February 20, 1959, showed no evidence of myasthenia gravis but of a progressive denervating lesion in the muscles of the upper limbs suggesting a myelopathy.

She was referred to Sir Russell Brain for a second opinion and was seen on March 13, 1959, when his report states in part: A rotatory nystagmus to right and left, bilateral weakness of soft palate, relative analgesia and anesthesia over the first and second divisions of the trigeminal nerve on both sides with sparing of the third, an unusually low hair line, minimal weakness in the arms, and an extensor plantar response on the right and possibly on the left. He thought the most likely explanation of these findings was some congenital abnormality of the foramen magnum, such as a basilar impression, an Arnold-Chiari lesion or



Fig. 2.—A basal view of skull (Case 3) to show encroachment along posterolateral margin of the foramen magnum on the left.

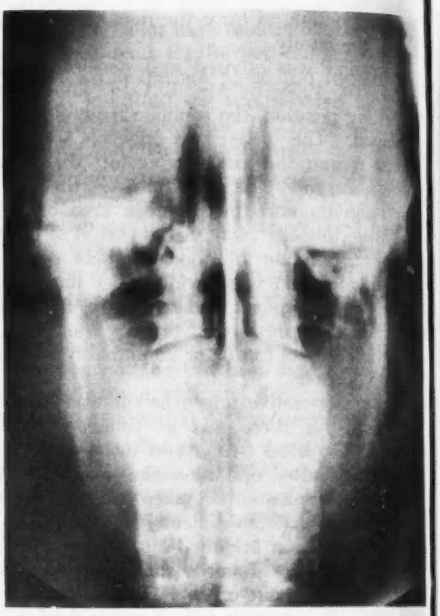


Fig. 4.—An anteroposterior tomogram of foramen magnum region to show the relationship of the dens and atlanto-axial joints to the tips of the mastoid processes (Case 3).

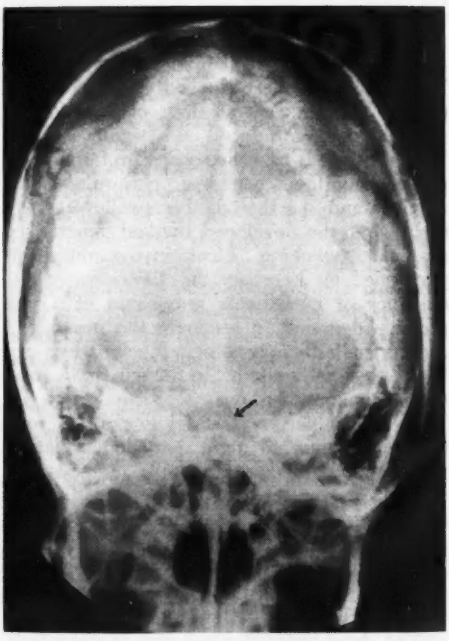


Fig. 3.—Towne's projection of skull (Case 3) to show encroachement along the left posterolateral margin of the foramen magnum.

both and he suggested the appropriate radiographic examinations.

These showed that the foramen magnum was extremely small and asymmetrical with a diagonal bony ridge cutting off the left posterolateral margin (Figs. 2 and 3). The posterior fossa was asymmetrical, the right being larger than the left. The left occipital condyle lay at a higher level than the right, and the slope of the left atlanto-axial joint was steeper than the right. The petrous apices were raised, and the left mastoid tip was higher than the right. The relevant measurements were: the dens and atlanto-occipital joint lay 15 mm. above Fischgold's line,[7] 10 mm. above McGregor's[12] and 8 mm. above Chamberlain's[4] (Fig. 4). The basal angle was 130°.

Air encephalography was performed on March 24, 1959, and showed evidence of tonsillar herniation; no ventricular filling was obtained. A ventriculogram was performed on March 26, 1959, using myodil, and it showed marked hydrocephalus with a long, narrow fourth ventricle, 4½ cm. long, situated at a lower level than normal, and below Twining's point; the aqueduct was in normal position and there was no evidence of a space-occupying lesion (Fig. 5). The contrast medium was held up in the fourth ventricle and a film taken

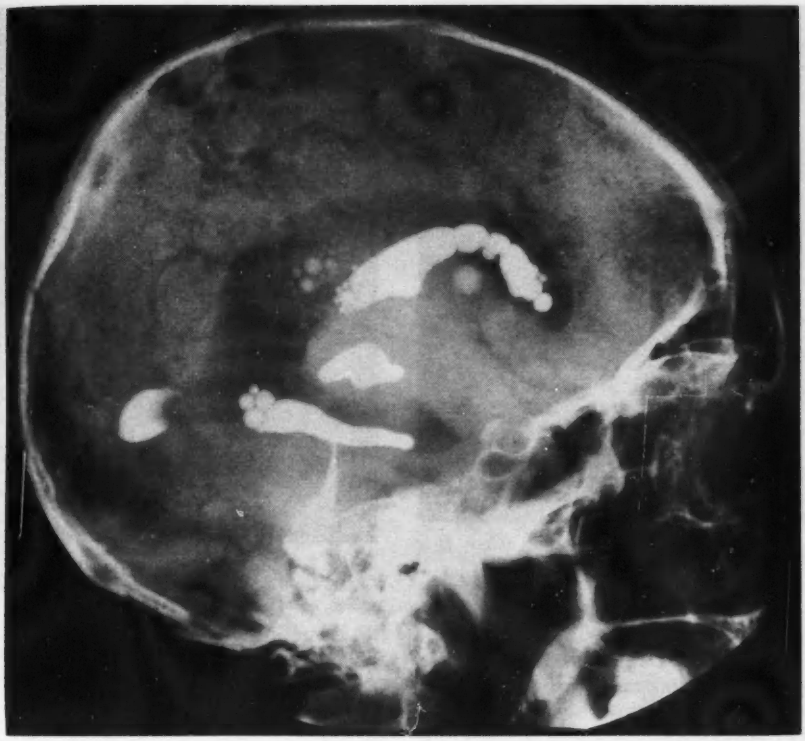


Fig. 5.—A myodil ventriculogram to show elongated fourth ventricle filled with air and myodil lying at a lower level than normal (Case 3).

two hours later still showed a considerable amount of myodil within it.

Operation was performed on March 31, 1959, by Mr. T. G. I. James. The plane of the squamous part of the occipital bone on each side of the foramen magnum inclined upwards with the foramen lying at a higher level. Because there was no space between the atlas and the occiput, removal of this bone was extremely difficult. The margins of the posterior aspect of the foramen magnum were thickened and curled upward into the cranial cavity.

The posterior aspect of the atlas and foramen magnum were removed and a band ⅛" thick was found lying immediately under the bone and forming part of the dura at this point. Interference with this band produced profound respiratory and cardiac upset. Her breathing became shallow and the pulse rapid and irregular but after five minutes recovery took place. With great care the band was divided and the dura which was now slack fell away.

The wound was closed in layers, but as the last stitches were being inserted the patient's heart stopped. Cardiac massage was performed within three minutes and a normal heart beat restored. The chest was closed and sealed drainage instituted. During the night tracheotomy was performed but she remained unconscious, in a state of spastic quadriplegia, and eventually died on July 12, 1959.

The limited postmortem examination was confined to the posterior fossa. The cerebellar tonsils were thin and elongated and ended below the level of the foramen magnum, and there was evidence of constriction of this part of the brain because below this level the cerebellum was thin and atrophic. There were dense arachnoid adhesions over the cerebellum and medulla which appeared smaller than normal but there were no syringobulbia or hydromyelia. The cervical spine nerves ran a normal course.

Histological examination showed extensive neuronal degeneration in the olives and in the nuclei of the floor of the fourth ventricle and demyelination of the spinocerebellar tracts.

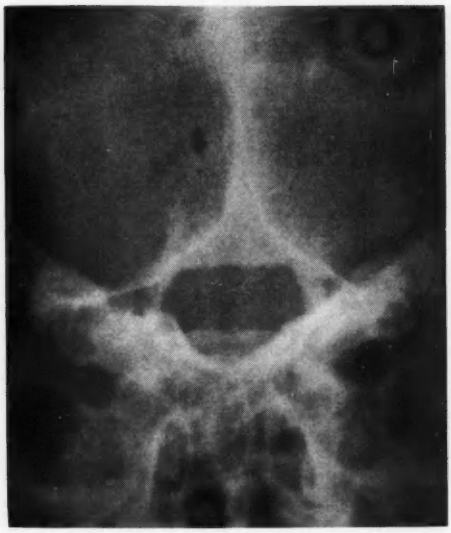


Fig. 6.—Towne's projection of skull (Case 4) to show abnormal foramen magnum.



Fig. 7.—Lateral view of cervical spine in flexion (Case 4) to show extension of upper cervical region.

CASE 4.—Lillian W., a 36-year-old former nurse, was admitted to the University of Alberta Hospital on December 22, 1960. Her principal complaint, present for about a year, was occasional blurring of vision which occurred intermittently and only in certain directions of gaze.

Three months before admission she had developed a stiff neck which was followed by rather severe pain behind the ears. This was associated with anorexia and nausea chiefly in the morning. Shortly after this she began to have occipital headaches and vertigo. She noticed jerking movements of the eyes when lying down, and in addition some unsteadiness of the feet with a tendency to veer to the left. A month before admission the patient had some difficulty in swallowing liquids and more recently solids.

On examination her speech was slightly slurred and had a nasal quality. The visual fields and fundi were normal. There was a very marked nystagmus with a rotatory element, more marked in the right eye and on right lateral gaze and it varied with the position of the head. The corneal reflex was diminished markedly on the left and slightly on the right; there was some hypalgesia on the left side of the face. All reflexes were increased and the plantars were equivocal.

Radiographs of the skull showed that the foramen magnum was unusually small and a bony ridge cut across the posterior aspect (Fig. 6). The posterior fossa itself was shallow and asymmetrical, the left side being smaller than the right. Flexion and extension studies of the cervical spine showed considerable restriction of movement at the atlanto-occipital joints with a tendency to hyperextension (Fig. 7). Tomograms of the region of the foramen magnum showed that the odontoid peg and the atlanto-axial joint lay at a much higher level than normal (Fig. 8). The dens lay 17 mm. above Fischgold's line,[7] 7 mm. above McGregor's[12] and 9 mm. above Chamberlain's[4] line. A posterior fossa encephalogram outlined the cerebellar tonsils in the upper cervical canal; no ventricular filling was obtained.

Operation was performed by Dr. T. J. Speakman on January 4, 1961, with the patient in the sitting position. Flexion of the head resulted in abnormalities of the respiratory rate and a loss of the pulse at the wrist; the head was therefore not flexed. The lower border of the foramen magnum, the arch of the atlas and the spinous process of the axis were crowded together, but this may have been due partly to position. The posterior rim of the foramen magnum including the inferior portion of the occipital bone was removed as widely as possible, then the posterior arch of the atlas and axis were removed.

The dura was opened in the midline and the cerebellar tonsils were found to descend almost to the upper border of the arch of the third cervical vertebra. There was no evidence

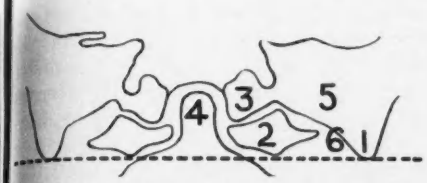


Fig. 8.—A sketch of anteroposterior tomogram to show the relationship of dens to the base of the skull (Case 4). (1) Mastoid process; (2) arch of atlas; (3) occipital condyle; (4) dens; (5) petrous pyramid; (6) digastric notch.

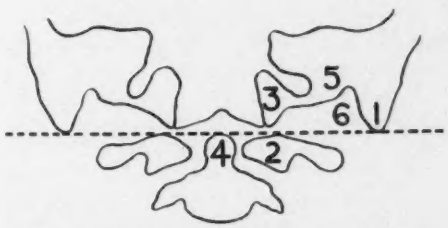


Fig. 9.—A sketch to illustrate the normal relationship of the atlas and axis to the base of the skull. This is a patient with an acoustic neuroma. (1) Mastoid process; (2) arch of atlas; (3) occipital condyle; (4) dens; (5) petrous pyramid; (6) digastric notch.

of a dural band. The dura was left open and the arachnoid was covered with Gelfoam.

When seen seven months after operation the patient was entirely free of symptoms; she was no longer ataxic, nor did she have any dysphagia.

Discussion

Basilar impression probably owes its first description, according to McGregor,[12] to Ackerman in 1790, and it was first diagnosed clinically by Schüller[16] in 1911. Etiologically, patients with this anomaly fall into two groups, primary and secondary. The secondary group, in which the deformity develops due to softening of the weight-bearing bones of the skull base as part of a skeletal disease, is not infrequently seen in advanced Paget's disease and cases have been described in osteogenesis imperfecta, rickets and osteomalacia. However, in the majority of cases there is no evidence of general skeletal disease and it is into this primary group that our cases fall.

The character of the deformity is essentially the same in both primary and secondary types, and O'Connell and Turner[15] have argued convincingly that mechanical factors alone in the presence of a congenital structural weakness of the skull base could reproduce all the features seen, including stenosis of the foramen magnum. One might account for the delayed and variable age of onset of symptoms, which is otherwise difficult to explain, on this basis, according to the rate of evolution. However, the deformity is accepted as developmental by most authors, and its frequent association with other malformations, such as atlanto-occipital fusion, failure of fusion of the posterior arch of the atlas, spina bifida, the Klippel-Feil syndrome and fusion of one or more cervical vertebrae favours this view.

Anatomy

A consideration of the embryology of the region is helpful in understanding how the deformity may arise. The basi-sphenoid is developed from the parachordal plate which extends from the foramen magnum to the pituitary fossa. The distal part of the parachordal cartilage includes the fused elements of three or four occipital sclerotomes. Cartilage extends on either side of the foramen magnum to form the occipital arches and unites behind it in the tectum posterium. The remainder of the parachordal cartilage ossifies to form the condylar part of the basi-occiput.

The occipital bone is formed from five parts, a pars basilaris, two partes laterales and an upper and lower part of the squama. At birth there are four pieces. The squama and partes laterales fuse between the first and fourth year, and the latter, with the pars basilaris, during the third to the sixth year. Incomplete assimilation of the most posterior of the three scleromeres, the proatlas, may result in the formation of an occipital vertebra.

While such an occurrence is said to be extremely rare, it is not uncommon to see "some formation or accentuation around the foramen magnum which may represent local vertebralisation of the occipital bone",[8] and such cases have been reported by Hadley[10] and List.[11] The anterolateral encroachment on the foramen magnum in Cases 1 and 2 and the posterior encroachment in Case 4 may be considered to have

arisen in this manner. The third patient's findings more likely represent a partial craniostenosis of the posterior fossa.

In a normal skull, the occipital condyles lie roughly in the plane of the mastoid processes, but in basilar invagination they are much higher (Figs. 8 and 9). In the latter condition the lips of the foramen magnum are turned up and infolded. The odontoid process lies far higher than usual and the available space in the foramen magnum becomes restricted and bean-like in shape. The petrous apices are elevated and may be asymmetrical.

A tight dural band is often found deep to the posterior arch of the atlas and may be more important in causing compression than the bony abnormality.[5, 9, 10, 17, 19] Such a band may be present in the absence of basilar impression or bony abnormality at the cranio-vertebral junction. O'Connell and Turner[15] found a narrowing of the dural theca at foramen magnum level in all their cases and advocate wide opening of the dura to provide a decompression. In two of our cases (Cases 2 and 3), a thick constricting band of dura was present and was divided. In Case 3 the dura was not opened because even gentle manipulation resulted in alarming changes in the respiratory and pulse rates. Although in Case 1 there appeared to be no constriction or narrowing of the dural theca, it is possible that dural decompression might have resulted in a more dramatic recovery. No band was present in Case 4 but the dura was left open.

A familial tendency has been stressed by Scoville and Sherman,[17] who reported its occurrence in two sisters aged 33 and 48 respectively. They also noticed that another patient had a nephew with syringomyelia, while the mother of another had a short neck and mildly unsteady gait. Bull, Nixon and Pratt[3] investigated all available relatives in their series of basilar invagination and found that a significant number of them had the same bony abnormality.

Our first two cases illustrate the familial tendency with the condition occurring most probably in three generations. There would seem little doubt that Margaret K's (Case 2) mother, who died at the early age of 49 after having suffered from progressive unsteadiness of the legs and epileptic fits, common symptoms of the disorder, has passed on the gene to her daughter, son and grandson. Whether this gene actually caused them to have an abnormally shaped posterior fossa at birth or merely a structurally weak skull base, which later became invaginated by weight bearing, is open to argument. There can, however, be no doubt about a genetic factor.

Clinical Features

Why neurological complications do not develop in some and at very variable ages in others, with apparently the same deformity, is still a question requiring a satisfactory answer. Symptoms precipitated by trauma have been reported in many instances, but there was no such history in our cases. The protean nature of the symptoms and clinical signs caused by basilar invagination makes diagnosis difficult and misdiagnosis common. It is often mistaken for disseminated sclerosis, syringomyelia, motor neurone disease and brain-stem or posterior fossa tumour.

McRae[14] analyzed the diagnosis of the referring physician in 68 of these cases and in almost half of them, the diagnosis was either multiple sclerosis or syringomyelia. The foramen magnum was thought to be implicated clinically in only nine cases, while the diagnosis of posterior fossa or brain-stem tumour was made in 11 patients. Considering the problem from another point of view, McRae[13] reported that he found bony abnormalities of the foramen magnum in 38% of all cases of syringobulbia and syringomyelia. This association has also been stressed by Spillane, Pallis and Jones,[18] who from their series concluded that "isolated basilar impression is usually asymptomatic, a syringomyelia syndrome is the commonest clinical concomitant".

Our cases, however, would support the impression obtained from the literature, that although isolated basilar impression may be asymptomatic, it may often cause neurological symptoms which are produced in varying combinations by compression of the upper cervical cord and brain-stem.

interference with lower cranial nerves or with the cerebrospinal fluid circulation.

Our first two patients presented what might be regarded as a "fairly typical" picture, if such can be maintained in a condition showing such diversity of signs. They both had symptoms and signs of progressive compression of the upper cervical cord, lower cranial nerve involvement and well-marked cerebellar signs. On the other hand, the third patient was most atypical. The presenting symptom was attacks of breathlessness and wheeziness of the chest, precipitated by exertion, nine months before any further symptoms developed. In retrospect these attacks were in all probability due to disturbance of the respiratory centre in the medulla from compression. This symptom, together with the striking fatigability of her voice, and inability to swallow, strongly suggested myasthenia gravis. Frank pyramidal and cerebellar signs developed late. Case 4 showed a similar symptomatology to Cases 1 and 2.

If the correct diagnosis is to be made in this condition it must be constantly borne in mind and the appropriate radiological investigations done in all cases with evidence of a high cervical or brain-stem disorder or symptoms suggestive of a posterior fossa space-occupying lesion.

Radiological Features

The radiological diagnosis of basilar impression is founded on a number of measurements. The best known of these, Chamberlain's[4] line, is drawn from the posterior margin of the hard palate to the posterior margin of the foramen magnum and is normally not more than 3 mm. below the tip of the odontoid peg. However, it is often difficult to identify the posterior lip of the foramen magnum and, as invagination progresses, it rises so that this measurement does not give a true index of the degree of invagination. In a case seen recently at autopsy where there was gross basilar invagination with flattening of the medulla against the dens, post-mortem radiographs showed only minimal abnormality using Chamberlain's method. The patient had been diagnosed during life as suffering from motor neurone disease.

McGregor,[12] recognizing the difficulty, proposed that instead the line should be drawn from the upper and posterior aspect of the hard palate to the most caudal aspect of the occipital bone. He recommends that the dens should not be accepted as being normal in position if it lies higher than 4.5 mm. above this line. Bull, Nixon and Pratt[3] relate the plane of the atlas to the plane of the hard palate and believe it to be a more reliable method of assessment. They state that an angle greater than 13° between the two planes indicates basilar impression and that an angle of more than 10° should be regarded with suspicion. Bull's angle may be normal, however, even in the presence of marked invagination as in Cases 3 and 4. Spillane, Pallis and Jones[18] believe that Bull's angle is partly determined by the degree of cervical lordosis present. All these methods relate the facial bones to the base of the skull and since these are developed independently, there remains a possible source of error. An unusually high or low palate or similarly a long or abnormally short dens or clivus may all lead to misdiagnosis.

However, for routine use McGregor's line appears to be satisfactory. All that is required is a true lateral film which is taken best with the patient in the prone position. Such a film shows the infolding of the foramen magnum well and an "off-lateral" film is even better.

For a demonstration of the abnormality at the base of the skull an anteroposterior view is best. This shows the relationship of the occipital condyles to the mastoid processes. Fischgold and Metzger[7] measured the height of the tip of the dens above a line drawn between the tips of the mastoid processes (Fig. 9). Normally this is not more than 2 mm. Their method gives a very clear-cut demonstration of the deformity, but tomography may be required. Spillane *et al.*[18] believe that anteroposterior tomograms of the foramen magnum are the only reliable way of demonstrating invagination. Myelography or air-myelography may be required to demonstrate prolapse of the cerebellar tonsils.

The relationship of platybasia to basilar invagination may be stated roughly as follows: in general, they are separate en-

tities. Platybasia was defined by Boogaard[1] as follows: the angle subtended by a line drawn from the frontonasal suture to the tuberculum sellae and from there to the anterior margin of the foramen magnum should not exceed 131.5°. Gross cases of acquired basilar impression may occasionally lead to some degree of platybasia by raising the anterior margin of the foramen magnum. However, in a recent case of Paget's disease, which showed the most gross invagination, there was no platybasia. The synonomous use of these terms should therefore be discarded. In none of the above cases was there any platybasia.

Surgical Treatment

In general, the results of surgical treatment fall into two categories, those leading to great improvement and those leading to arrest of an otherwise progressive condition. Cases 1, 2 and 4 illustrate the latter category very well. O'Connell and Turner[15] stress the importance of operative treatment as soon as possible after neurological signs and symptoms have developed. They feel that with earlier diagnosis and treatment the severe disability which may develop can be prevented. It seems, however, that in those cases with bulbar symptoms or hydrocephalus the operative risk is very great. Our third case falls into this group.

Stauffer[19] reported 11 cases treated surgically and stated that of these, nine improved. All had dural constricting bands, four of these showed no evidence of basilar invagination. Five cases with tonsillar herniation and two without improved dramatically.

McRae[13] discussed 21 patients of whom 10 had signs and symptoms attributable to the deformity; five of these were secondary to Paget's disease. All 10 showed evidence of basilar invagination, and three had herniation of the cerebellar tonsils. Some improved after operation while the others showed no progression of signs and symptoms. McRae[13] considered the finding of a convex clivus to be of significance in the 12 cases in which it was found.

Scoville and Sherman[17] reported 10 cases and concluded that surgical decompression arrests, but only occasionally improves the neurological disability. Gustafson and Oldberg[9] reported five cases and stated that the foramen magnum is usually small and misshapen and often eccentric. Two of their cases improved, one showed slight improvement, another improved and then died; autopsy in this case showed the medulla to be constricted. Another case with an eccentric foramen magnum and a restricted anteroposterior diameter also died. These cases with lateral encroachment of the foramen magnum are identical with Case 3 in the present series.

It is seen from these reports that while operation may be necessary for the arrest of an otherwise progressive condition, dramatic results can only be expected occasionally and that the mortality rate is high.

Summary

Four cases of bony abnormality at the foramen magnum are reported. Two of these occurred in a mother and son and the familial tendency is remarked upon. Two further cases are reported to illustrate difficulties in the diagnosis of this syndrome. The clinical features, the radiological diagnosis and the surgical results are discussed.

It is concluded that recourse to surgery may be necessary but dramatic relief of signs and symptoms cannot always be expected. In this series one patient improved dramatically, one patient showed no progression, a third showed symptomatic improvement and one died. These results are in keeping with those reported by other authors.

It is a pleasure to express our appreciation to Dr. F. Pygott for his help and encouragement in the preparation of this paper, and to Dr. James Bull and Dr. T. J. Speakman for their helpful advice and criticism. We should also like to thank Dr. G. Monckton for permission to include Case 4. Mr. Maxwell Ellis for referring Case 3 and the Photographic Departments of the Central Middlesex and the University of Alberta Hospitals for the reproduction of radiographs. We also thank Mrs. B. Rhind for the sketches.

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Résumé

Dans les déformations de la base du crâne étudiées ici (os occipital) il y a lieu de distinguer deux groupes: un groupe dit primaire et un groupe secondaire. Dans le groupe secondaire rentrent les cas consécutifs à des maladies osseuses généalisées: ostéomalacie, ostéogénèse imparfaite, maladie de Paget. Le groupe primaire comprend les cas d'étiologie inconnue; cependant, des considérations embryologiques et peut-être des facteurs héréditaires peuvent-ils aider à en interpréter la pathogénie. L'os occipital est formé par la soudure de cinq parties: une partie basale, deux parties latérales et deux parties écailleuses, inférieure et supérieure. Une absence de soudure en arrière peut aboutir à la formation d'une vertèbre occipitale; cette éventualité est rare mais on rencontre assez fréquemment des cas où il y a un épaississement du bord du trou occipital pouvant être interprété comme une vertébralisation partielle de l'os occipital.

Dans l'investigation de la base, les condyles occipitaux se trouvent dans un plan situé beaucoup plus haut que celui des apophyses mastoïdes: les bords du trou occipital sont éversés en haut; l'espace disponible dans le trou occipital devient très réduit. Souvent, dans ces cas on rencontre un épaississement fibreux de la dure mère qui vient encore rétrécir le tout.

L'aspect clinique de la difformité est variable. Ceci rend souvent le diagnostic difficile: la confusion avec la syringomyélie et la sclérose en plaques est fréquente. Le diagnostic devra être fait radiologiquement. avec les incidences appropriées; la myélographie permettra de mettre en évidence une hernie des amygdales.

Le traitement est chirurgical: on peut ainsi arriver à une grande amélioration ou tout au moins arrêter un processus progressif. Cependant, dans les cas avec symptômes bulbaires ou hydrocéphalie, il semble que le risque opératoire soit trop grand.

Les auteurs présentent ensuite l'observation de quatre malades. Deux d'entre eux étaient familiaux: une femme de 56 ans et son fils de 19 ans. En ce qui concerne les résultats, il y eu un cas de très bonne guérison, un cas de stabilisation, un cas sans amélioration et un cas de mort.

CASE REPORTS

SPLENIC ANEURYSM*

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SPLENIC aneurysm has been described, chiefly as a source of unexpected lethal intraperitoneal hemorrhage, for almost 200 years.[3] However, the antecedent clinical picture, so seldom referred to in standard texts, deserves greater familiarity because of the recent development of a safe method of abdominal aortography, using an intravenous injection of contrast media.[5]

It is the most common intra-abdominal aneurysm except for that of the aorta. Unlike all other aneurysms it is much more common in females,[3] and tends to rupture particularly during late pregnancy. We wish to report one case with typical dyspepsia cured by surgical intervention before hemorrhage occurred.

CASE REPORT

The patient, a 49-year-old white married woman, gave an unusual history of three years of almost unrelenting nausea and vague epigastric distress, relieved by vomiting but aggravated by eating or lying down. As a consequence it was often worse at night and associated then with frontal headache. Despite these symptoms her weight remained fairly constant, and no hunger dyspepsia or episodic variation had occurred. At the beginning of her illness she was examined elsewhere. A gastric analysis was stated to show achlorhydria, and after gastroscopy her illness was diagnosed as atrophic gastritis. Glutamic acid medication produced no relief. Shortly afterwards, during hysterectomy for intractable menorrhagia, her stomach was palpated but no abnormality was noted.

On subsequent physical examination she appeared thin, was acutely sensitive in the epigastrium, and complained of nausea with even the lightest of pressure. No other abnormalities were detected and auscultation of the abdomen was not carried out. However, an upper gastrointestinal barium series showed a persistent indentation in the greater curvature of the stomach (Fig. 1), suggesting deformity due to pressure from an extrinsic mass. She was admitted to hospital and there a vague mass was detected under the left costal arch but respiratory shift could not be demonstrated. Laboratory investigation showed — hemoglobin 13.2 g. %, a leukocyte count of 3500 per c.mm., with polymorphs 47%, metamyelocytes 2%, lymphocytes 50% and monocytes 1%. Apart from this neutropenia no abnormalities were seen in the blood film to suggest compensated chronic hemolysis or platelet reduction. The sedimentation rate was 6 mm. in the first hour and gastric analysis showed hypochlorhydria to histamine stimulation.

On laparotomy an enlarged spleen was found, and a hard but pulsatile aneurysmal mass was detected in its hilum. The remaining viscera appeared healthy and the splenic artery with its aneurysm and spleen were



Fig. 1.—A persistent extrinsic filling defect is present in the proximal part of the greater curvature opposite the spleen. No intrinsic abnormalities of the stomach are demonstrated.

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resected (Fig. 2). The fixed spleen measured 13½ x 6 x 8 cm., and showed only passive venous congestion on histological examination. The tortuous supple splenic artery, 11 cm. in length, showed atheromatous streaking only, and 2 cm. from its termination an aneurysm, 1.5 cm. in diameter, could be seen arising from one half of its circumference. On opening the aneurysmal wall, it was 0.1 cm. thick with a patchy eggshell-like calcification and a rough intimal surface largely covered by laminated adherent fibrin thrombus. Microscopically, the wall of the aneurysm was composed of a very thin layer of fibrous tissue with patchy calcification of the attached intima. Near the mouth of the aneurysm the splenic artery wall showed severe atheromatous changes which replaced the fibroelastic coat for several millimetres. Multiple sections of the remainder of the artery at intervals showed a completely intact fibroelastic coat and minimal atheroma only. The thrombus in the aneurysm was of a bland type and showed minimal organization. The splenic vein was buckled by the aneurysm, but showed no other changes. After operation the platelet count rose from 260,000 per c.mm. to 775,000 per c.mm. by the fourth postoperative day. No spontaneous thromboses occurred and the platelet count returned quickly to normal. The patient's complaints have been completely relieved in the six months of follow-up study, her first relief since the illness began.

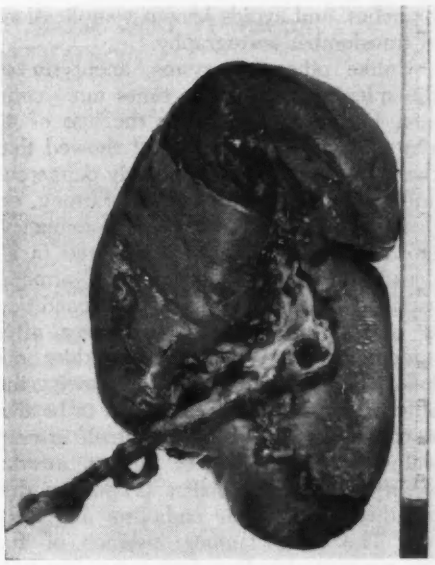


Fig. 2.—The saccular aneurysm of splenic artery is partly filled by thrombus. It has been opened and rotated from the hilum where it buckled the splenic vein.

Discussion

The earlier clinical reports of splenic aneurysm dealt almost exclusively with the terminal phase of rupture, especially during pregnancy. Deterioration was usually far too rapid to allow surgical intervention, but attention was focused on about 12% of patients in whom a two-stage rupture occurred with blood leaking first into the splenic pedicle and retroperitoneum, and later bursting into the lesser or greater sac.

However, persistent symptoms often have preceded rupture of the aneurysm by a period of as long as 14 years. These symptoms are usually those of nausea or persistent abdominal discomfort when the stomach is filled with food, and are aggravated characteristically by lying down or eating, and eased by vomiting. Epigastric or left upper quadrant pain is sometimes produced by stooping, or by the postural shifts of a full stomach as in our case, but radiation of the pain is rare. Most of the symptoms are produced by congestive splenomegaly and this organ was palpable in one half of the cases reviewed by Owens and Coffey.[3] The palpation of a pulsatile aneurysm, rarely more than one inch in diameter, or detection of an audible localized bruit over it has been quite uncommon. It should be noted that extensive calcification of the normally tortuous splenic artery is very common in the elderly, and this alone may produce a thrill, bruit or systolic murmur without the presence of an aneurysm.

Roentgenography now provides the most valuable confirmatory information in many cases, for apart from demonstration of gastric distortion due to splenomegaly, the aneurysm itself may be shown in a variety of ways. These include pulsatile filling defect in the stomach wall, a circumscribed opacity or concentric rings of opacity due to calcification in the sac wall itself. Contrast visualization of splenic and hepatic arteries by intravenous abdominal aortography as devised by Steinberg[5] now appears to be the safest mode of establishing the diagnosis of aneurysm in these

branches, and avoids known complications of translumbar aortography.

Unlike other aneurysms, aneurysm of the splenic artery is five times more common in the female below the age of 45 years. Sheehan and Falkiner[4] showed that these aneurysms were extremely dangerous and liable to rupture without warning, especially during the latter part of pregnancy. However, they have been reported in all decades. While the majority were formerly ascribed to arteriosclerosis this condition is commonplace in splenic artery after middle age and its incidence does not follow that of aneurysm. On reviewing the older literature most cases seem to be due to congenital defects in the splenic arterial wall, as in the case of hepatic arterial aneurysm, with a smaller proportion due to atheroma, mycotic embolism and trauma. The most famous instance of the latter occurred in President James Garfield, who died two months after being shot in the abdomen by an assassin from rupture of a splenic aneurysm of traumatic type. The aneurysmal change seems to appear especially when the arterial flow is increased as a consequence of splenomegaly. It has been reported in conditions as diverse as malaria and Gaucher's disease.

The risk of rupture in all these cases, especially in late pregnancy and whether associated with splenomegaly or not, makes operative resection imperative whenever the condition is discovered.

Summary

The postural dyspepsia associated with aneurysm of splenic artery is described and methods of diagnosis briefly reviewed. We report one case cured by surgical intervention before hemorrhage occurred.

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Résumé

C'est l'histoire d'un cas d'une femme âgée de 49 ans souffrant de nausées et de douleurs épigastriques vagues depuis une période de trois ans. Ces symptômes sont aggravés par la position couchée et par l'ingestion d'aliments. A l'examen on note que la sensibilité épigastrique est très forte. Le transit baryté montre une indentation large et persistante dans la grande courbure de l'estomac. Une laparotomie exploratrice permet de trouver une rate considérablement augmentée de volume avec un anévrysme pulsatile dans son hile. La rate et son pédicule furent enlevés. Les suites opératoires furent sans histoire.

La paroi de l'anévrysme présentait un épaississement considérable avec des placards d'athéromatose.

Il est à noter que contrairement aux autres types d'anévrysmes, les anévrysmes de l'artère splénique sont particulièrement fréquents chez les femmes. Les ruptures spontanées peuvent se voir surtout lors de la grossesse; dans ces cas, l'hémorragie est généralement trop rapide et trop surprenante pour qu'il soit possible de faire quoi que ce soit d'utile. La radiographie est évidemment le meilleur moyen diagnostic; outre les déformations que l'on peut apercevoir sur les viscères creux, il est possible de mettre en évidence l'anévrysme lui-même par l'aortographie avec injection d'un moyen de contraste.

TWO SYNCHRONOUS PRIMARY MALIGNANT TUMOURS (KIDNEY AND COLON)*

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THE following case was a baffling diagnostic problem and not until operation was the true state of affairs recognized.

CASE REPORT

M.M., a 56-year-old trapper, first noticed gross blood in the urine in December 1959 while voiding in the snows of northern Alberta. This hematuria was painless and intermittent. In January 1960 he noticed some constipation, but was able to get his bowels to move with mineral oil. There was no blood in the feces. His illness became acute on December 6, 1960, when he had lower abdominal cramps, distension and difficulty in getting rid of flatus. Stoical for a whole year about his hematuria and change of bowel habit, he was forced finally to see his doctor, was admitted to the local hospital on December 10, 1960, and transferred to the University of Alberta Hospital on December 13, 1960, one week after his acute symptoms appeared. He had passed no gas per rectum for 24 hours before admission to the hospital. From the time of his initial symptoms in December 1959 until his admission to hospital in December 1960, he had lost 25 lb. in weight, dropping from 155 to 130 lb.

On admission his condition was surprisingly good, with normal temperature and pulse rate, and fair hydration. When asked to pass urine, he jumped nimbly off the bed and freely voided 8 oz. of grossly bloody urine, which he said was similar to that which stained the snow.

His abdomen was grossly distended, tympanitic, and one could see and hear the peristaltic rushes, which occurred at the same time as his lower abdominal cramps. Abdominal tenderness and rebound tenderness were absent, and no masses were palpated at first. Rectal examination revealed no abnormality; the rectum was empty of feces, and a sigmoidoscope was inserted freely for 8 in. in a patient who climbed on to the Buie table with aplomb despite a large distended abdomen.

Microscopic urinalysis confirmed the presence of many red blood cells. A flat plate of the abdomen (Fig. 1) showed numerous dilated loops of small and large bowel with thickened peritoneum between them. There were some streaky calcifications on the right side, the significance of which was not realized. An emergency barium enema showed a suggestion of a tumour in the sigmoid colon (Fig. 2). Further investigation showed a hemoglobin of 14.1 g., white blood cell count 5300/c.mm., normal blood urea nitrogen and serum electrolytes, and a normal radiograph of the chest.

Initial treatment consisted of the insertion of a long intestinal tube for decompression and the administration of intravenous fluids. Nothing was given by mouth. A Cantor tube was passed through the pylorus and into the upper jejunum on December 16. As his distension partially subsided, a very large firm mass could be palpated in the region of the right kidney. He began to pass a little gas per rectum and had a small bowel movement. However, his abdomen remained markedly distended, peristaltic waves could be seen, and peristaltic rushes were heard. On December 16, on retrograde pyelography, the right kidney was seen to be grossly distorted, with dye shown only high up in the region of the upper pole. The impression was that of a massive renal tumour involving the major part of the kidney. The calcification noted on the original plain film (Fig. 1) was thought to be calcification in a hypernephroma. We were unable to correlate a diagnosis of hypernephroma with his obvious clinical intestinal obstruction, but postulated that he might have had some unusual type of metastatic involvement or perhaps some internal hernia. There were no external hernias, and he had no scars on his abdomen from previous operations.

Operation was undertaken finally on December 19, 1960; the abdomen was opened through a right split-rectus incision centering on the umbilicus. There was no free intraperitoneal fluid. There was a large mass in the right retroperitoneal area, extending from the dome of the diaphragm to the right iliac crest. This mass was very firm, somewhat nodular, and seemed to have some mobility up and down but not transversely because of its size.

*From the Departments of Surgery and Pathology, University of Alberta, and the University of Alberta Hospital, Edmonton.

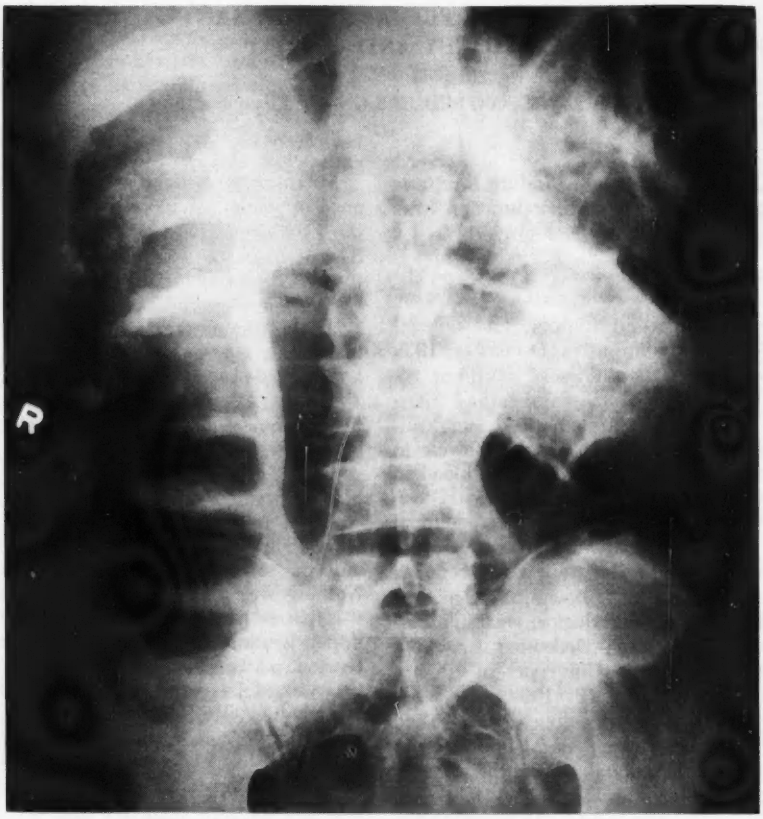


Fig. 1.—Flat plate abdomen showing dilated loops of small and large bowel and the suggestion of calcification in right kidney region.

There was considerable dilatation of small and large bowel as far distally as the sigmoid colon, where, in the mid-sigmoid region, a freely movable, stenosing, napkin-ring tumour, having all the characteristics of a primary malignancy of large bowel, could be palpated. The liver, stomach, spleen and left kidney were normal to palpation.

A right nephrectomy was decided upon, the skin incision extending up to the right costal margin, because of the size of the tumour. The peritoneum in the right gutter lateral to the ascending colon was incised over the length of the retroperitoneal tumour, and the dissection carried medially to expose the renal pedicle. After division of the renal artery and vein, it was noted that there was no tumour spread in the latter, and after division of the ureter, the huge mass was rather easily mobilized and removed intact. Because of the large bowel obstruction, a simple rod right transverse colostomy was performed and the bowel was brought out through the same rectus incision. The abdomen was closed around the colostomy.

The colostomy was opened in 48 hours and the Cantor tube removed in 72 hours. Convalescence was uneventful and in three weeks a sigmoid resection was performed.

On January 9, 1961, the abdomen was opened through a transverse left rectus cutting incision in the lower abdomen. Again there was no free fluid in the peritoneal cavity, the liver was grossly free of metastases and no other tumours could be palpated in the colon. In the mid-sigmoid region, there was a stenotic, napkin-ring tumour, which measured 5 cm. in diameter and completely encircled the bowel, but was mobile. Several enlarged nodes could be palpated in the mesentery. The bowel above the tumour was moderately dilated, but in good condition and well prepared. Tapes were placed around the bowel above and below the tumour, and a

wide resection of a very mobile and redundant sigmoid colon and its mesentery was carried out, with end-to-end anastomosis.

Postoperative course was again uneventful. A barium enema carried out on January 23, 1961, showed normal function at the anastomosis, with no leakage. On January 25, 1961, the colostomy was finally closed intraperitoneally and resected with an end-to-end anastomosis 16 days after the sigmoid resection and five weeks after nephrectomy and transverse colostomy. He was discharged from hospital on February 4, 1961, in good condition with his wounds healed.



Fig. 2.—Barium enema showing a probable tumour in sigmoid colon.

Pathological Reports

Right kidney (Fig. 3).—"This is a 1200 g. mass measuring 14 cm. in diameter, having a thick greyish-white capsule. Spread across one surface is a remnant of a kidney, measuring 2 x 4 cm. with fairly normal architecture. Cross sections through the tumour itself show many large areas of necrotic, reddish-brown tissue, associated with cavitation and calcification. A lobular pattern is given to it by strands of firm, almost cartilaginous tissue coursing through the tumour. Other areas are composed of soft, yellowish-red tissue. Microscopically (Fig. 4), sections through the large kidney tumour show the histological features of hypernephroma. In the central portion of the tumour there are large areas of fibrosis and calcification."

Diagnosis: Hypernephroma—kidney.



Fig. 3.—The right kidney showing hypernephroma (note the rim of normal kidney tissue and the huge well-encapsulated tumour).

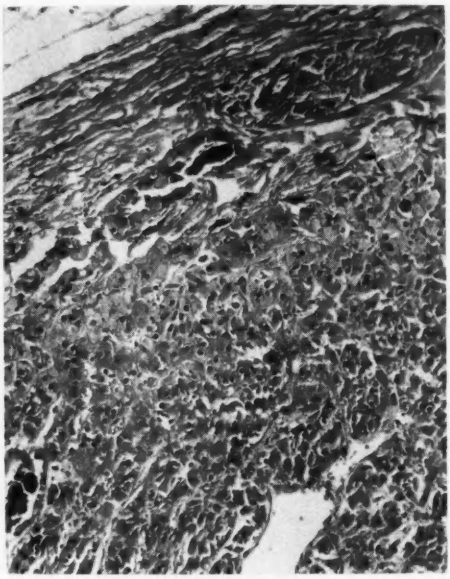


Fig. 4.—Photomicrograph. Hypernephroma (renal cell carcinoma).

Colon (Fig. 5).—"After formalin fixation, this is a 26-cm. length of sigmoid colon. At 11 cm. from one end, the serosa is puckered throughout its circumference by a hard tumour mass. On opening the bowel, at 9 cm. from the other end, there is a 5 x 5 cm. heaped-up ulcerating tumour mass, greyish-white in colour with hard rolled edges, completely encircling the bowel and narrowing the lumen to pin-point dimensions. Attached to the bowel there is a wide segment of mesentery containing multiple firm lymph nodes, the largest 1 cm. in diameter, and the smallest 2 mm. Twelve of these nodes were taken for microscopic section. Histologically (Fig. 6), sections through the tumour mass show the features of a rapidly growing invasive adenocarcinoma which penetrates the bowel almost to the serosal surface. Sections through the 12 regional lymph nodes were negative for tumour metastases."

Diagnosis: Adenocarcinoma—Grade III—sigmoid colon. Lymph nodes—regional (12), negative for metastases.

Discussion

Multiple primary maligant tumours have interested surgeons since Billroth described the first case almost 100 years ago. Warren and Gates,[2] in an exhaustive review of the world literature in 1932, concluded that multiple malignant tumours occur more frequently (3.9% of all malignant cases) than can be explained on the

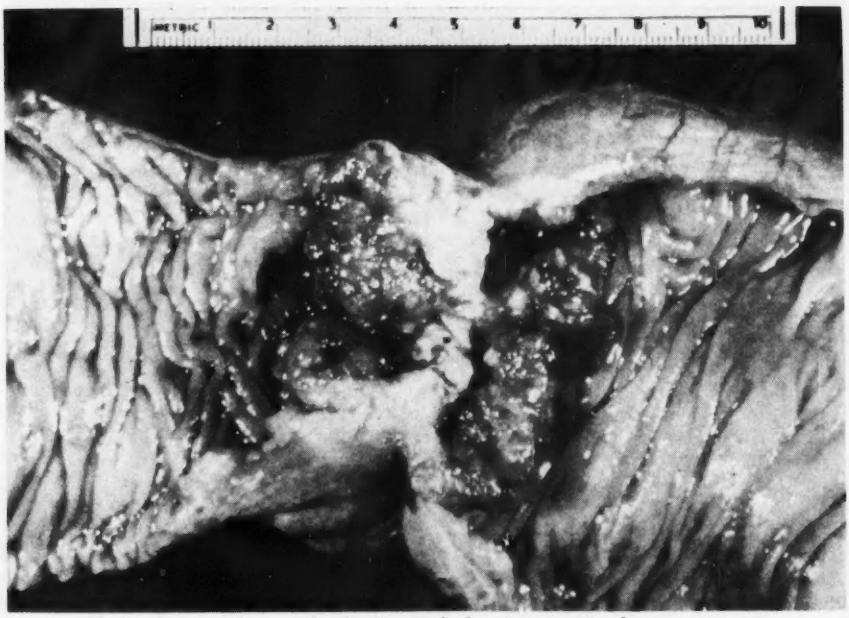


Fig. 5.—Portion of sigmoid colon (opened) showing stenotic ulcerating tumour.

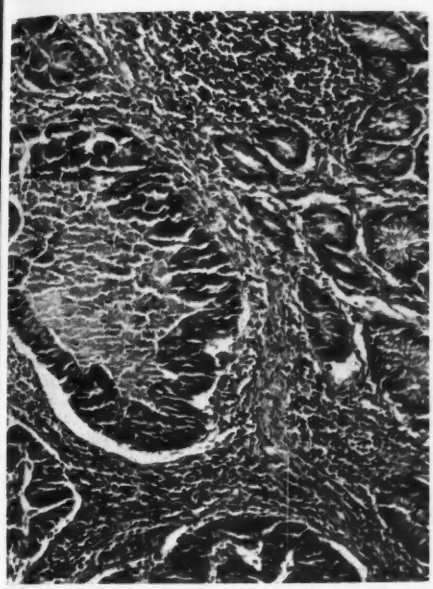


Fig. 6.—Photomicrograph. Adenocarcinoma of sigmoid colon.

basis of chance. Thus a patient who has one cancer is more susceptible, is more likely to get a second cancer, or has an inherited predisposition to cancer. Their statistics were based mainly on autopsy cases. The following rules were laid down as criteria for the diagnosis for multiple primary malignancy: (1) Each of the tumours must present a definite picture of malignancy; (2) each tumour must be distinct, and (3) the probability of one being the metastasis of the other must be excluded.

Slaughter,[3] reviewing the world literature up to 1944, found only 14 patients with this combination of primary malignancy of kidney and colon, and since then only three more instances could be found. Most of these cases appear to have presented at different times; that is, they were metachronous and not synchronous as in this case. Watson,[4] reviewing 16,626 cases of malignancy in Saskatchewan over many years, found the combination of hypernephroma with carcinoma of the rectum (not with colon) only once. This makes our case, presumably the eighteenth to be reported in the world literature, and presenting synchronously, very rare indeed.

The tendency is for multiple tumours to occur in the same type of tissue, either in the same organ or paired organs. Thus in the collective review of Slaughter,[3] 40% of the multiple tumours were of the same organ and 14.4% in paired organs. The common sites for multiple tumours of one organ were in order, skin, colon and stomach. By far the most common site for multiple tumours of paired organs was the breast, then the testicle and the ovary. Ovarian double primary malignancies may be much more common than testicular, but they are difficult to prove. Multiple primary malignant tumours of the gastrointestinal tract are a fairly common combination—the stomach and colon, stomach and esophagus, colon and esophagus; and the combination of cancer of the breast and the female genital tract is not uncommon.

Slaughter[3] agreed with Warren and Gates[2] that the incidence of multiple malignancy in patients with cancer was greater than could be explained by mere chance. However, Watson,[4] although agreeing that some patients have a marked tendency to develop multiple cancer of the same organ, i.e. skin, colon and bladder, found in a large well-followed series that the incidence of multiple cancer was 3.3%, which was no different from the incidence of single malignancies in the normal population. He concluded that having one cancer neither confers immunity nor predisposes the patient to the development of another cancer.

Summary

A case of two primary malignancies of kidney and colon presenting simultaneously is described. This particular combination of multiple primaries is rare indeed. Only 17 cases have been reported in the literature, and these were chiefly in autopsy material.

Multiple primary malignant tumours tend to appear in the same organ or paired organs, such as the breast. Except for multiple primary malignancies in the skin and

in the colon, and possibly in the breast, they are probably no more common than one would expect from chance alone. Thus, having one cancer, neither predisposes to nor safeguards against the development of cancer in another organ.

However, multiple primary malignancies are sufficiently common (4% in all cases of malignancy) that one should realize that a second tumour is not necessarily a metastasis from the first, and should be treated hopefully.

In this patient the prognosis must be guarded. If the five-year survival rate for hypernephroma is 33% and for sigmoid carcinoma 50%, then he has approximately an 18% chance of living for five years.

The author appreciates the support of the Department of Pathology, University of Alberta, and its chief, Professor John W. Macgregor, in the preparation of this article. Dr. Macgregor is one of those pathologists who takes much interest in the problems of the surgeon and thus is able to be of particular assistance to him.

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Résumé

Présentation du cas d'un homme de 56 ans hospitalisé en décembre 1960, à l'Hôpital de l'Université d'Alberta. Il souffrait d'hématurie depuis un an et d'une légère constipation chronique. A l'admission son état général était satisfaisant. L'abdomen était enflé et faisait entendre du tympanisme à la percussion. L'examen rectal était négatif, de même que la sigmoïdoscopie. L'urine contenait une grosse quantité de sang. L'examen radiographique à vide de l'abdomen montrait de nombreuses anses grêles et grosses dilatées.

Le traitement fut d'abord conservateur: on introduisit une sonde à aspiration continue et l'on mit le malade à la diète totale. Ceci permit à la distension abdominale de régresser et l'on put alors palper une masse ferme dans la région du rein droit. Le diagnostic posé cliniquement à ce moment fut celui de tumeur rénale avec métastase abdominale.

Le patient fut opéré; on découvrit une grosse tumeur rétropéritonéale occupant l'espace compris entre le dôme diaphragmatique droit et la crête iliaque du même côté. Cette masse était dure et nodulaire. D'autre part il existait également une tumeur annulaire située sur le côlon sigmoïde dans sa partie moyenne. On ne trouva aucune autre lésion. On pratiqua alors une néphrectomie droite et une colostomie de décharge. Cette première intervention fut bien supportée et l'on put dans un deuxième temps, un mois plus tard, faire l'ablation de la tumeur sigmoïdienne, suivie d'une anastomose bout-à-bout. Les suites opératoires furent sans histoire et le patient put quitter l'hôpital après une quinzaine de jours.

Il s'agissait d'un hypernéphrome du rein droit et d'un adénocarcinome du troisième degré du côlon sigmoïde. L'anatomie pathologique ne permit pas de trouver de signes de métastases ganglionnaires.

Cette combinaison de deux cancers est rare. Le pronostic dans ce cas peut être évalué de la façon suivante; étant donné que l'indice de survie de cinq ans est de 33% pour l'hypernéphrome et de 50% pour le carcinome sigmoïdien, il reste à ce malade 18% de chance de ne pas faire de récidive.

HEPATIC LOBECTOMY IN INFANCY FOR HEPATOBLASTOMA

"Primary liver tumours in children are extremely uncommon. Packard and Palmer report an incidence of nine cases in 126,000 admissions to a children's hospital. They also note that the majority of liver tumours in children are malignant. Gross, too, out of 18 cases of hepatic tumour found 13 to be malignant . . .

"Most liver tumours in children occur below the age of two years. Steiner, in a review of 77 cases of primary liver cancer in children, found 41 which developed before the age of two years . . . In Bigelow and Wright's series of 95 cases, 55 were under two years old. Although the sex incidence differs in various series, it appears that malignant liver tumours are more common in male children.

"A hepatoblastoma in an infant aged seven months is reported. The tumour was excised and after four years there has been no recurrence. It appears to be the first long-term survival of hepatoblastoma in the literature."

—Borman, J. B., Harbott, A. J. and Morris, D.: *Brit. J. Surg.*, **49:** 11, 1961.

MESENCHYMOMA IN THE RETROPUBIC SPACE*

CLARENCE SCHNEIDERMAN, M.D., MORRIS A. SIMON, M.D. and MORRIE M. GELFAND, M.D., *Montreal*

MESENCHYMOMA, or the mixed mesodermal tumour, has been reported in various areas of the body, and the urogenital tract[1-4] has been a frequent site of involvement by both the benign and malignant varieties. Other sites recorded for the tumour are muscle and subcutaneous tissue, mesentery of the bowel,[5] and the breast. Stout[6] has defined the mesenchymoma as a tumour which is presumably formed from the cellular derivatives of mesenchyme, and is composed of cell masses more or less resembling the various derivatives of the supportive and reticuloendothelial systems.

The case reported here is of great interest because of the involvement of the bladder, urethra and vagina by a large tumour situated in the retropubic space, which caused vesical neck obstruction and dystocia. The surgical approach to this lesion, which presented vaginally, was through the retropubic space, rather than through an anterior vaginal route. Many extensive paravaginal and periurethral lesions can be more readily dealt with through this route than by a vaginal approach.

CASE REPORT

A 21-year-old white housewife, pregnant for the first time and with an estimated date of confinement on January 20, 1959, was admitted in early labour on January 25, 1959. The prenatal course had been entirely uneventful. On pelvic examination a mass was found approximately 10 cm. in diameter which filled the vagina, chiefly in its anterior aspect (Fig. 1). The vertex of the fetal head was floating, being prevented from engaging by this tumour. A cesarean section was performed during which it was noted that the tumour, situated in the retropubic space, was firm and irregular in shape. The patient's postoperative course was uneventful and she was discharged on the twelfth day following the cesarean section.

The patient was seen six weeks postpartum, and on examination when the vaginal tumour was palpated, it seemed to have increased in size and had become somewhat cystic. On April 13, 1959, she was admitted to hospital. There were no symptoms of pain due to urinary obstruction, infection or hematuria. Urinalysis revealed a specific gravity of 1.020, acid reaction, a trace of albumin, and on microscopical examination an occasional red blood cell, many white blood cells per high power field and a few epithelial cells. The hemoglobin was 90% (14.0 g.); the white blood count was 5400 per c.mm., with a normal differential, and the sedimentation rate was 3

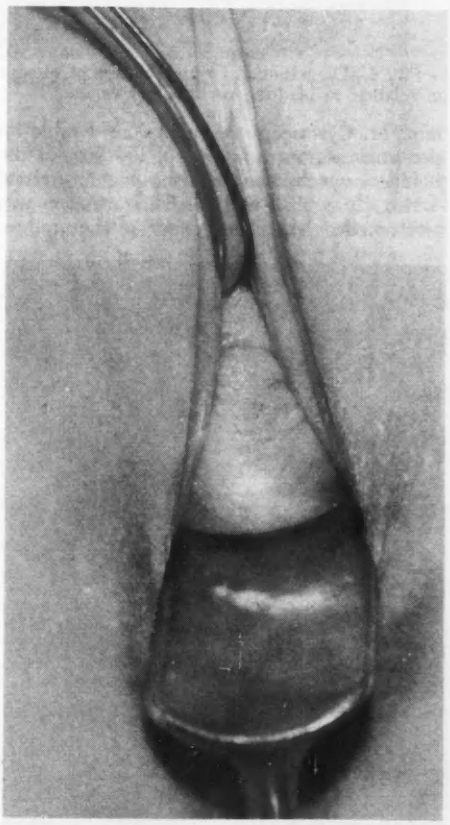


Fig. 1.—Vulvar view showing retractor on posterior wall of vagina, with a large mass projecting into the anterior vaginal wall. (Ureteral catheters protrude from the urethral orifice.)

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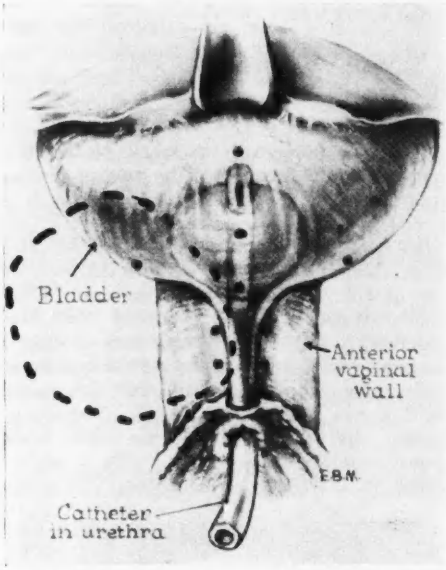


Fig. 2.—Diagrammatic representation of tumour in relation to bladder, urethra and vagina.

mm./hr. Cystoscopy revealed considerable indentation of the right side of the floor of the bladder which elevated the right ureteral orifice (Figs. 2, 3 and 4). This intrusion was also noted on the right portion of the urethra.



Fig. 3.—Cystogram (AP view) showing compression effect on the right lateral wall and neck of bladder and urethra by the tumour.



Fig. 4.—Cystogram (oblique view) showing indentation of the bladder wall by tumour.

Catheters, French #5, were easily passed to both renal pelves.

On April 15, 1959, the retropubic mass was excised through a transverse lower abdominal incision above the symphysis pubis, incising the anterior rectus sheath and separating the rectus muscles in the midline. The bladder was brought into view and retracted posteriorly exposing the retropubic space. Upon inspection of that area, a large mass which appeared to be beneath the endopelvic fascia was seen adjacent to the right side of the vesical neck and urethra. A dissection was carried out of the large tumour which invaginated the bladder wall, vesical neck and right aspect of the urethra (Fig. 5). The upper portion of the mass was solid but the lower portion was of a cystic consistency. Because of its location and cystic character the possibility of a Gaertner's duct cyst was considered. After the mass was elevated the anterior vaginal wall was incised and then closed. Drains were placed in the retropubic space and the abdomen closed. The postoperative course was essentially uncomplicated and the patient was discharged 11 days after the operation. She has been examined periodically since that time and two years later there is no evidence of a recurrence of the tumour.

Pathology

The specimen consisted of a globular fragment of firm, grey-pink tissue measuring 10



Fig. 5.—View of operative area, with tumour mass dissected free, except for its attachment to urethra and vesical neck.

x 9 x 4 cm. Its outer surface was wrinkled and covered by fibrous adhesions. Sectioning showed an irregular grey-white edematous surface and a cystic space measuring 2.5 cm. in diameter (Fig. 6). Anatomical orientation was not possible. Several sections were submitted for histological examination.

Upon microscopical examination, multiple sections taken through the large tumour mass showed it to be limited along the periphery by compact tissue, presumably indicating its encapsulated nature. The tumour itself was composed of relatively dense fibrous connective tissue in which numerous spaces lined with endothelium were present; most of these were empty but some contained small numbers of red blood cells and some precipitated albuminous material. Many of these spaces consisted of single layers of endothelial cells only (Fig. 7), while other vascular spaces showed thick, concentrically arranged inner muscular and outer fibrous coats. Throughout the tissue, most of which was hyalinized, there were irregular groups of spindle-shaped cells cut in various planes (Fig. 8). Some of these had blunt nuclei, and superficially resembled fibroblasts. In many situations the supporting tissue was markedly edematous and, indeed cystic degeneration was seen in a number of areas. No interstitial hemorrhage and no inflammatory infiltrate was seen.

Sections stained by the trichrome method revealed that much of the tissue took a deep blue stain, including the outer walls of the majority of the thickened blood vessels. However in a number of areas some of the spindle-shaped cells, cut at multiple planes, showed red-staining cytoplasm, indicating their smooth muscle nature. Careful examination of these cells failed to reveal any transverse striations in any situation.

Diagnosis.—Benign mesenchymoma (angioleiomyofibroma).

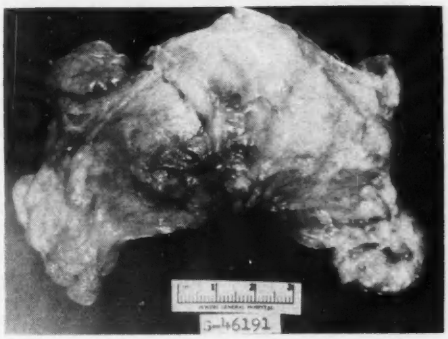


Fig. 6.—The gross specimen of tumour after its removal.



Fig. 7.—Low power view to demonstrate collagenized connective tissue and angiomatous structures.

Discussion

Mesenchymomas are tumours composed of a mixture of tissues all of which are derived from mesenchyme. Thus, such tumours may be composed of admixtures of any of the following tissues in varying ratios: fibrous, adipose, smooth muscle, skeletal muscle, vascular structures, endothelium, cartilage, bone and myxomatous tissue. Such tumours are in all probability dysontogenetic growths.

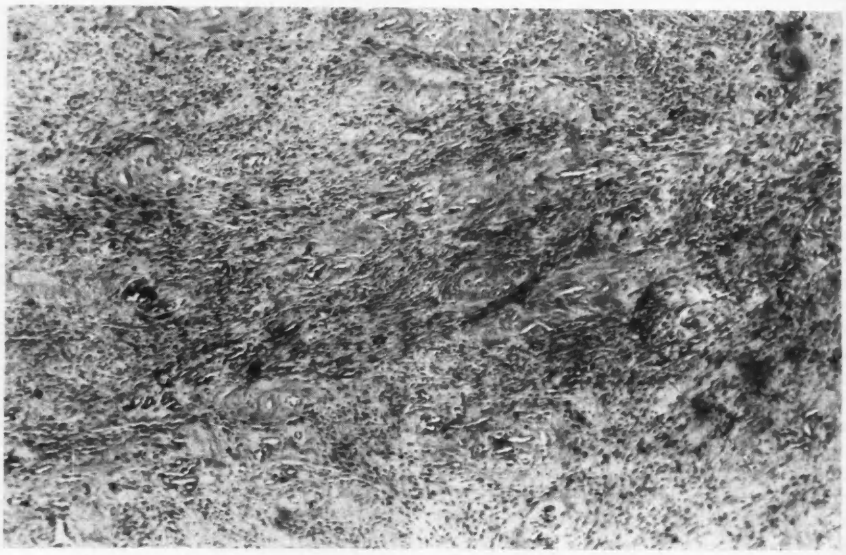


Fig. 8.—A relatively low-powered view showing spindle-shaped cells. Many of these were shown to be smooth muscle after staining with Masson trichrome stain.

Because of the presence of at least three mesenchymal derivatives, namely, fibrous connective tissue, vascular endothelium and smooth muscle, the term "mesenchymoma" is applicable to the tumour reported in this communication. Both benign and malignant forms of mesenchymomas occur,[7] but the pathological features of the present case indicate that this tumour is benign.

Operative approach to this large mass presenting vaginally was possible through the anterior vaginal wall or the retropubic space. The latter was selected because of our experience with the retropubic exposure in the urethrovesical suspension operation (Marshall Marchetti) for stress incontinence, where dissection can be easily carried out down to the most distal segment of the urethra. This has prompted us to use this avenue for the surgical exposure of large paravaginal or paraurethral lesions, because they can be dealt with more easily in this manner than by the vaginal approach.

Summary

A case of mesenchymoma (angioleiomyofibroma) arising in the retropubic space and involving the bladder, urethra and vagina has been presented. Attention is drawn to the facility with which large paravaginal and paraurethral lesions are exposed through the retropubic approach.

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Résumé

La tumeur mixte d'origine mésodermique, encore appelée mésenchymome, peut se rencontrer dans n'importe quelle localisation. Un cas est présenté ici avec localisation rétropubienne.

Il s'agit d'une femme de 21 ans admise à l'Hôpital Général Israélite de Montréal pour grossesse à terme, au début des premières douleurs d'accouchement. A l'examen, on trouve que l'engagement de la tête de l'enfant est gêné par une masse tumorale d'un diamètre de 10 cm. environ qui obstrue le vagin sur sa face antérieure. L'accouchement est alors effectué par césarienne. Après cette première intervention, la malade quitte l'hôpital sans complication. Elle est réhospitalisée six semaines plus tard et il semble à ce moment que la tumeur vaginale a augmenté de volume. Une intervention pour ablation est pratiquée: on trouve alors cette tumeur envahissant l'espace rétropubien, la paroi vésicale antérieure et la portion droite de l'urètre. Les suites opératoires furent excellentes. Le diagnostic histopathologique est celui de; angioleiomyofibrome, mésenchymome bénin.

Volume 4 of the *Canadian Journal of Surgery* contains five issues, from October 1960 to October 1961 inclusive. All future volumes of the Journal will be published within the calendar year, the first issue appearing in January and the other issues in April, July and October. This change has been made to comply with the wishes of subscribers.

THYMIC CYSTS OF THE NECK*

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Among the malformations of the neck, an interesting and rare entity is the thymic cyst. According to Arey,[1] the thymus originates at the end of the sixth week from ventral sacculations of the third pharyngeal pouches. Hollow at first, these two diverticula become solid rapidly by epithelialization. By the eighth week, their lower ends enlarge and become united. These unified ends attached to the pericardium descend into the thorax and reach their normal position in the anterior mediastinum. If a portion of either of the superior ends persists in the cervical region, it may develop as heterotopic thymic tissue and give rise to a solid tumour or form a cyst.

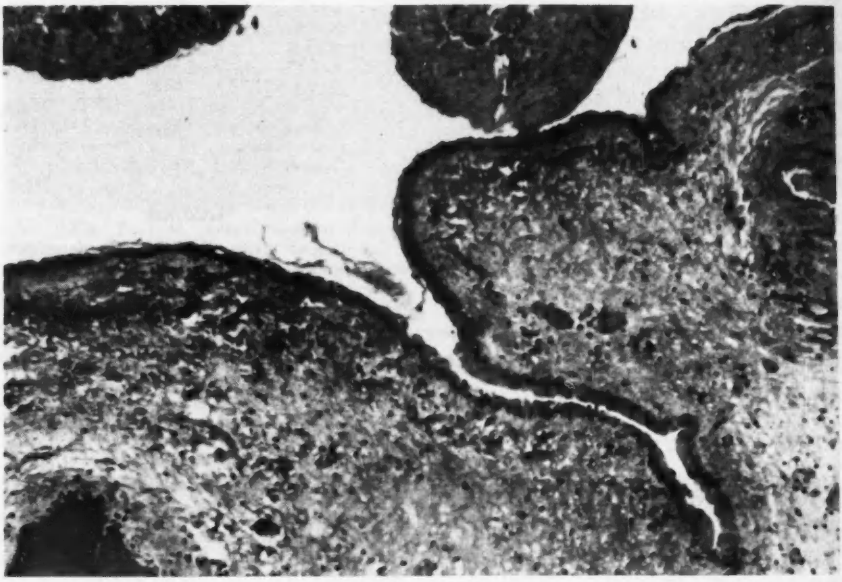


Fig. 1.—Lumen of the cyst limited by a fibrous wall covered with a cylindrical epithelium.

Case Report

A 6-year-old girl was admitted to hospital on February 22, 1960, because of a right cervical lump which was first noticed by her mother six days before admission. There was no serious illness in the child's past history and on physical exmination a soft, painless, fluctuating mass was palpated under the anterior border of the right sternomastoid muscle that extended from the clavicle up to the hyoid bone. A radiograph of the chest was normal.

An aspiration biopsy was performed, and on cytological examination the 15 c.c. of brownish fluid contained polymorphonuclear leukocytes, histiocytes, squamous cells and cholesterol crystals. After the patient was treated for an upper respiratory infection, surgery was performed under general anesthesia. The mass was found to be a multilocular cyst, located within the carotid sheath between the common carotid artery and the internal jugular vein. The upper pole was deep behind the carotid bifurcation and the lower end extended down into the thorax. The postoperative course was uneventful, and the patient was discharged on March 15, 1960.

Macroscopic examination revealed a multilocular cyst filled with an amorphous brownish

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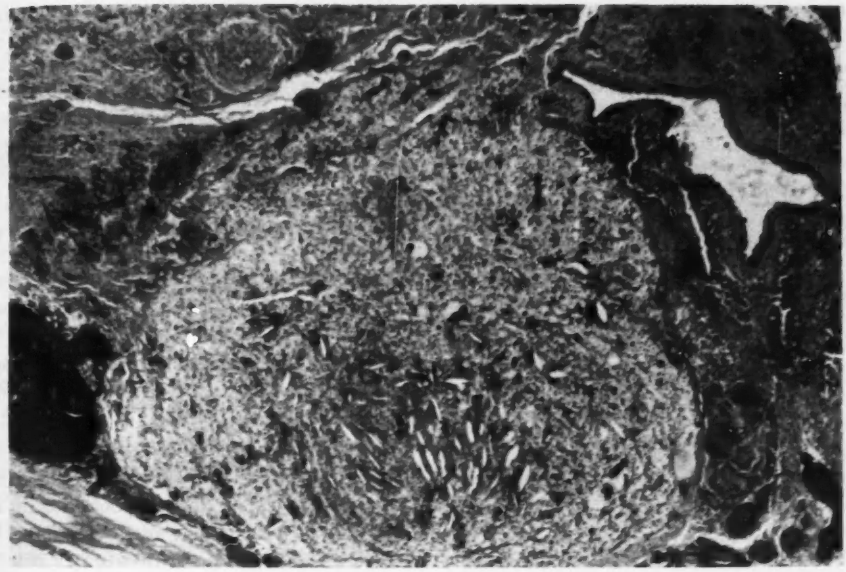


Fig. 2.—To the right: lumen of the cyst. Centre: lipophagic granuloma with cholesterol crystals. To the left: the black nodule is thymic tissue.

material. The thickness of its wall varied from a few millimetres to 10 mm. On histological examination, the cystic spaces contained acidophilic granular material and a few cholesterol crystals. The wall, covered by a stratified squamous epithelium which was cylindrical in some areas (Fig. 1), was made up of connective tissue in which there were some chol-

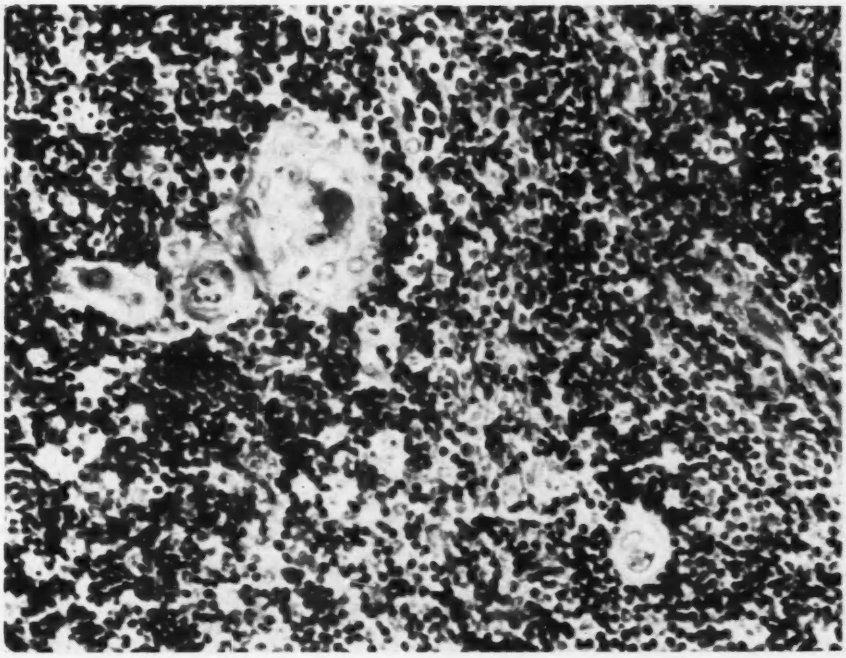


Fig. 3.—High power of a thymic nodule showing Hassall's body.

TABLE I.—Thymic Cysts of the Neck: Clinical Data

Author	Number of cases	Year	Sex	Age	Duration	Location
Pollosson and Piéry[8]	1	1901	M	1½	—	Right neck.
Pezcoller[7]	1	1929	F	5	—	Suprasternal notch and mediastinum.
Hyde *et al.*[4]	1	1944	M	5	8 months	High in right anterior cervical triangle.
King[5]	8	1949	—	—	—	—
Heinz[3]	1	1950	M	6	—	Cervical.
Weller *et al.*[11]	1	1951	F	4½	4½ months	Left anterior and posterior cervical triangles.
Williams and Gerber[12]	1	1957	M	6	Several weeks	Anterior to the left sternomastoid muscle.
Crawford *et al.*[2]	1	1957	M	6	3 years	Lateral to right lobe of thyroid.
Viar *et al.*[10]	1	1959	M	5	4 years	Along lower anterior border of left sternomastoid.

esterol granulomas (Fig. 2) and many foci of thymic tissue (Fig. 3) with typical Hassall bodies (Dr. Robert Garneau).

Review of the Literature

The instances of thymic cyst of the neck described in the literature have been summarized in Tables I and II. The first was published in 1901 by Pollosson and Piéry,[8] who reported the tumour as a malignant thymic cyst. Unfortunately, their patient died on the day of the operation.

Pezcoller's case,[7] reported in 1929, was interesting because the tumour presented in the midline, above the sternal notch. In 1944, Hyde, Sellers and Owen[4] reported the first successful excision of a thymic cyst.

In 1949, King[5] reported on 76 branchial cysts and extensive histological studies revealed that eight of them contained thymic tissue in their walls. Several other instances of thymic cysts have been reported since then. Finally, an extensive review, including all thymic cysts both mediastinal or cervical, was made by Krech, Storey and Umiker,[6] in which they reported four additional cases but none of these were situated in the neck.

Discussion

Our case is similar to those reported in the literature. The age of the patient and clinical data are comparable. A laterally placed fluctuating mass in the neck which does not move with swallowing should make one think of this diagnosis, particularly in a child. An anteroposterior radiograph of the chest is necessary to determine the degree of extension of the mass into the mediastinum.

The pathological features are usually those of a multilocular cyst which contains a brown fluid with cholesterol crystals. The lining epithelium is variable; the wall contains granulomata and fibrous tissue; the

TABLE II.—Thymic Cysts of the Neck: Pathological Data

Author	Contents	Lining
Pollosson and Piéry[8]	Multilocular with brown fluid.	Flat pavement cells.
Pezcoller[7]	Brown fluid with granular debris	Cuboidal and cylindrical cells.
Hyde *et al.*[4]	Multilocular with yellowish-brown liquid containing cholesterol crystals.	Flat cells.
King[5]	—	—
Heinz[3]	—	Squamous epithelium.
Weller *et al.*[11]	Unilocular, brown fluid.	Squamous epithelium and flat cells.
Williams and Gerber[12]	Unilocular, yellow fluid containing cholesterol crystals.	Flat pavement cells.
Crawford *et al.*[2]	Multilocular, brown red fluid.	Stratified squamous epithelium.
Viar *et al.*[10]	—	Squamous epithelium.

finding of thymic tissue in the wall makes the diagnosis.

The frequency of malignant change in these cysts is difficult to establish, but malignant thymomas have been said to have originated in aberrant thymic tissue.

The surgical removal of the cyst is not usually difficult. The danger of removing one or more parathyroid glands with the mass is real, but since these cysts are unilateral, serious consequences should be minimal.

Finally, what is the nature of such a cyst? Speer[9] has suggested five possible ways in which a cyst may be formed within the thymus: (1) from embryonal remnants; (2) from sequestration of involuted areas; (3) from degenerating Hassall's corpuscles; (4) from lymph vessels, and (5) from neoplastic processes. However, considering the embryology of the neck, the location of the cyst and the findings of King,[5] the lesion could be simply a branchial cyst with thymic tissue in its walls.

Summary

The medical literature on thymic cysts arising in the neck has been reviewed and a case reported. The clinical and pathological data have been discussed and the pertinent features of these tumours have been examined in some detail.

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Résumé

On sait que le thymus se forme à la fin de la sixième semaine embryonnaire à partir des tissus de la région antérieure de la troisième poche pharyngienne. Ces bourgeons sont attachés au péricarde et lors du développement de ce dernier, sont entraînés dans la cavité thoracique. Si quelques fragments subsistent dans la région cervicale, ils peuvent donner naissance à des kystes ou à des tumeurs solides.

Un cas de ce genre, chez une petite fille de six ans est présenté. Il s'agissait d'une tuméfaction molle et fluctuante située sur la face latérale du cou, immobile lors de la déglutition. Une radiographie antéro-postérieure du thorax devra toujours être faite pour déterminer l'extension de la tumeur dans le médiastin.

Du point de vue anatomie pathologique il s'agit d'une masse kystique multiloculaire contenant un liquide brunâtre et des cristaux de cholestérol. Les parois sont généralement bordées d'un épithélium d'aspect variable; la présence de plages de tissu thymique permet le diagnostic. Il existe certainement une possibilité de dégénérescence maligne, bien que cette question soit peu connue.

Le traitement consistera en l'excision de la masse. Il faudra toujours penser au risque d'enlever en même temps une ou plusieurs parathyroïdes par accident; cette éventualité serait cependant peu grave puisque ces tumeurs sont toujours unilatérales.

RUPTURE OF AORTIC ANEURYSM INTO DUODENUM: A SUCCESSFULLY TREATED CASE*

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THE rupture of an aortic aneurysm into the lumen of the gastrointestinal tract is an uncommon cause of hematemesis and melena. Nevertheless, reports of such cases are appearing in the literature with increasing regularity. In a recent report,[1] 63 cases were collected in which the presenting symptoms were referable to such a condition.

Voyles and Moretz,[2] in analyzing such cases in the literature, found that only 27% died within the first six hours following the sudden rupture, and concluded that approximately 60% of such occurrences are amenable to surgical exploration and treatment, provided a diagnosis can be made without delay. They also reported a single case in which the diagnosis was established preoperatively and in which successful replacement of the aneurysm with a teflon-ivalon graft was carried out. Unfortunately, this patient developed a leak at the site of the anastomosis on the 34th postoperative day, which required surgical repair, and he succumbed five days later with clinical findings suggestive of a cerebrovascular accident. To the best of our knowledge, this is the only reported case to date in which an aortic aneurysm, eroding into bowel and presenting as massive gastrointestinal hemorrhage, has been successfully resected.

With the increasing application of replacement procedures for abdominal aortic aneurysms, the relatively long-term complication of rupture of the graft into the gastrointestinal tract is now appearing in the literature. A recent review of this subject by Cordell, Wright and Johnston[3] reveals that rupture of five aortic homografts into the duodenum has been successfully treated by resection and replacement with plastic prostheses, while seven cases of rupture were treated without success. Rupture of the suture line between vessel and prosthesis with hemorrhage into the gastrointestinal tract has been reported nine times; two of these patients were successfully treated by surgical excision and further replacement.

It is the purpose of this communication to report what is believed to be the first wholly successful surgical management of rupture of an abdominal aortic aneurysm into the gastrointestinal lumen.

CASE REPORT

Mrs. D.B., a 33-year-old woman, was admitted to the Royal Alexandra Hospital on June 11, 1957, with the complaint of severe bilateral lumbar pain of seven days' duration and some pain in the right groin and buttocks of two days' duration. The onset of these symptoms was heralded by recurrent chills and fever. Examination at the time of admission showed only minimal tenderness in the right groin which was associated with movement of the right leg. There was neither costovertebral tenderness nor bladder tenderness. Pelvic examination was normal. Initial treatment was appropriate to the diagnosis of low back syndrome and consisted of bed rest, antispasmodics and analgesics. However, the symptoms failed to respond to this regimen and shortly after admission the patient's temperature rose to 102.4° F. and continued at this elevation for four days. During this febrile episode, her white blood count was 19,650 with 95% polymorphonuclear neutrophils; an occasional white cell per high power field was found during urinalysis. The sedimentation rate was elevated to 43 mm./hr. A urine culture was reported as negative and the blood urea nitrogen was within normal limits. The therapy was changed; antibiotics and salicylates were administered with rapid improvement and complete subsidence of symptoms. Subsequent investigation, including radiographs of the lumbosacral spine and an intravenous pyelogram, was normal. A flat plate of the abdomen was also reported as normal. A diagnosis of pelvic inflammatory disease was entertained but this was not borne out by gynecological examination. Consequently, the final diagnosis at time of discharge was back pain of undetermined origin.

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This woman was readmitted subsequently to the same hospital on July 25, 1958. A history was obtained of severe mid-lumbar back pain occurring the day preceding her admission to hospital, followed by a fainting episode. The back pain continued for 24 hours and was followed by massive melena per rectum. At the time of admission the patient was in a moderate degree of shock and was passing bright red liquid blood per rectum. A sigmoidoscopic examination was carried out but this failed to reveal any pathology in the lower colon. During this time there was nothing abnormal noted on abdominal examination. She experienced two bouts of vomiting; the initial emesis was bile-coloured and not bloody, the second was blood clot. During the night she suffered hemorrhage per rectum, which was almost exsanguinating and required 2500 c.c. of whole blood and 1000 c.c. of polyanhydroglucose (Dextran) to bring under control. The bleeding appeared to stop with these measures and her blood pressure, pulse and hemoglobin returned to normal limits. A complete gastrointestinal investigation was carried out in the ensuing days. This included a barium swallow for esophageal varices, radiographs of the stomach and duodenum for peptic ulceration, and a barium enema with air contrast for pathology of the colon; all were reported as negative. A small bowel "follow-through" was done and reported as showing no evidence of pathology. Because of the massive gastrointestinal hemorrhage which this woman had experienced, an exploratory laparotomy was performed seven days after admission, and six days after the clinical arrest of hemorrhage. The only positive finding was an aneurysm of the abdominal aorta which arose just below the left renal artery and extended down to involve both common iliac arteries. This aneurysm had eroded the posterior wall of the fourth part of the duodenum (Fig. 1). The duodenum in this area was stretched tensely over the mass and on opening the anterior duodenal wall, a 1-mm. perforation of the posterior wall was encountered which was plugged by a small thrombus. The duodenotomy and the abdominal incision were closed and arrangements were made to carry out a resection of the abdominal aorta shortly after. The abdomen was reopened six hours after the initial laparotomy; the aneurysm and a portion of the fourth part of the duodenum were resected and an orlon-knit bifurcation graft was inserted to re-establish vascular continuity. The patient was ambulant within 48 hours and experienced no postoperative complications. She was discharged

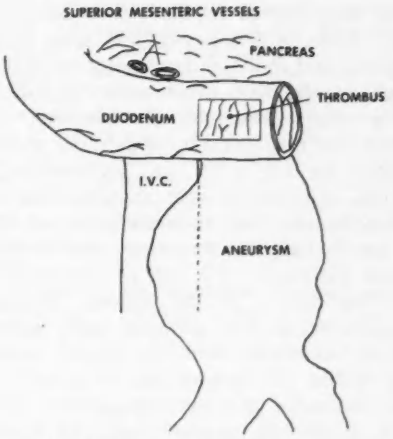


Fig. 1.—Sketch of findings at laparotomy. A window has been removed from the anterior wall of the fourth part of the duodenum to expose the area of erosion and thrombosis.

13 days after the aortic graft was performed. The pathological report described atheromatous plaques in the excised portion with aneurysmal dilatation at several sites. Microscopical examination showed a small round cell infiltrate, mainly lymphocytes and plasma cells, lying in the adventitia of the vessel. The only other investigation carried out at this time included serological determinations, the Kahn and Wassermann reactions, which were negative.

The patient has now been followed up for 30 months since her aortic resection and grafting procedure and has remained well throughout this time. Femoral pulsations are normal at the present time and she has no complaints.

Discussion

It is interesting to speculate about the cause of this patient's previous admission to hospital with a complaint of severe back pain. This probably represented an initial minor leak of the aneurysm into the retroperitoneal space. If this is true, the fact that she carried on, asymptomatic, for a further 13 months is notable. Even in retrospect, review of the radiographs of the abdomen taken during her first admission gave no hint of the presence of an aneurysm. The exhaustive radiographic investigation undertaken before laparotomy, even though the diagnosis of an aneurysm was not entertained clinically, shows no evidence suggestive of an aortic aneurysm.

The diagnosis of rupture of an aneurysm as a cause of this patient's melena, hematemesis and shock, did not enter our minds. The investigative procedures carried out after the cessation of bleeding necessitated considerable abdominal manipulation on the x-ray and sigmoidoscopic tables; the risks of such manipulation are a frightening thing to contemplate in light of the findings at laparotomy, and bring to mind the simile of a keg of dynamite. In another review of this subject, Skromak *et al.*[4] report that patients with rupture of an aneurysm into the bowel usually die within 10 days of one or a series of such episodes. It is interesting that a small clot in the aneurysmal wall can control aortic pressures for such a period, despite extensive abdominal manipulation.

Our preoperative diagnosis at the time of laparotomy was an undiagnosed duodenal ulcer or a bleeding Meckel's diverticulum. However, with severe bleeding of an obscure nature, we were prepared to find almost any rare gastrointestinal abnormality, with the exception of the one that was found. Considering that most reported cases of this condition occur in elderly patients and that over 50% of such aneurysms can be diagnosed by radiological examination and by abdominal palpation,[5] there is some justification for our failure to consider this condition in our differential diagnosis of this 34-year-old woman preoperatively.

The rarity of arteriosclerotic aneurysms in patients in this age group, coupled with the fact that the aorta proximal to the aneurysm appeared normal to palpation and inspection aroused the suspicion of some other etiological factor as a cause for this aneurysm. Tuberculous lymphadenitis with subsequent erosion into aorta and the subsequent formation of a false aneurysm has been followed by erosion into the gastrointestinal tract and has been cited as a rare cause of massive hematemesis and melena,[5] but no such findings were noted at laparotomy in this case. Syphilis has always been considered an etiological factor in many aortic aneurysms. The negative serology in this case, together with no history of infection, tend to rule out this diagnosis. The round cell infiltration noted by the pathologist, however, is characteristic of luetic infection. Consequently, a *Treponema pallidum* immobilization (T.P.I.) test was done and reported as negative on two occasions. Therefore, the final diagnosis must be an arteriosclerotic aortic aneurysm.

This patient has recently been examined by one of us, two years and four months following the successful surgery. Nothing was found to indicate failure or degeneration of the graft. The peripheral pulses are equal and full in both lower extremities and no abnormality can be detected on abdominal examination.

Inasmuch as most operations for aortic replacement, using either a homograft or plastic prosthesis, are performed upon elderly people whose life expectancy is limited, we feel that we have an excellent opportunity in this case to observe the fate of such a graft over a long period of time. To date we have relied on clinical findings, and have not carried out any direct examination by aortography. In view of the rupture of grafts into the gastrointestinal tract alluded to in the introduction,[3] any further gastrointestinal hemorrhage in this patient will be viewed as a surgical emergency.

Summary

The successful treatment of a patient with rupture of an abdominal aortic aneurysm into the duodenum has been reported. In the light of our experience, such a diagnosis should be considered in all obscure cases of severe gastrointestinal bleeding.

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Résumé

Ceci est l'histoire d'un cas d'anévrysme de l'aorte abdominale ayant rupturé dans le duodénum et qui put être traité avec succès.

Il s'agit d'une femme de 34 ans admise à l'hôpital "Royal Alexandra" en 1958. Elle se plaignait de douleurs lombo-sacrées, de lipothymies et de méléna depuis quelques jours. A l'admission, la malade était en état de léger choc et rejetait du sang rouge par le rectum. La sigmoïdoscopie ne montra cependant rien de spécial. Des crises de vomissements survinrent, dont l'un était composé de caillots de sang. Il devint nécessaire de la transfuser (2500 ml. de sang et 1000 ml. de Dextran). Après avoir fait un transit qui ne montra rien de spécial, on se résolut à une laparotomie exploratrice. On découvrit un anévrysme de l'aorte abdominale s'étendant de l'artère rénale gauche jusqu'à la bifurcation iliaque. Cet anévrysme avait érodé la paroi postérieure du duodénum dans sa quatrième portion et il existait une communication entre le vaisseau et la lumière duodénale, communication qui ne se trouvait obstruée que par un petit caillot. Le duodénum fut fermé et l'on procéda à une résection de l'aorte abdominale, suivie de greffe par un tube d'orlon. Les suites opératoires furent bonnes et la malade est maintenant guérie.

Du point de vue étiologique, il a été possible d'éliminer la syphillis dans ce cas. Il s'agissait d'un anévrysme d'origine athéromateuse.

CORONARY ENDARTERECTOMY*

"The feasibility of coronary endarterectomy in some patients with segmental occlusive disease has recently been demonstrated by Longmire and Bailey. Success of this procedure, however, depends upon proper selection of patients with localized segmental occlusions and the perfection of techniques of small vessel surgery. Recent development of improved methods of coronary arteriography, including cine-roentgenography, have made possible the localization of segmental occlusions with reasonable accuracy, thus facilitating the selection of patients for operation. Once the diagnostic problems have been solved, the need for better techniques of arterial disobliteration and reconstruction will become increasingly important. At present, thrombosis of the vessels after thromboendarterectomy is the principal cause for both early and late failures. In a recent study relating to small vessel surgery we demonstrated that stenosis of the lumen after closure of an arteriotomy was a common cause of thrombosis. To avoid this complication, a technique was described wherein an oval-shaped patch of suitable material was sutured to the wound edges, preventing luminal constriction. This method was successfully applied to coronary and femoral vessels in experimental animals. The following study consists of a more intensive investigation of the application of this technique to coronary artery surgery. A comparison of various types of patch materials was made in experimental animals, and the feasibility of the clinical application of this method was evaluated in human hearts removed at autopsy in which segmental coronary occlusions were present . . .

"This study consists of an evaluation of the patch technique as an adjunct to coronary endarterectomy. Patches made from arterial autograft, venous autograft, pericardial sac, and Dacron prosthesis were successfully used to facilitate closure of longitudinal incisions in the circumflex branch of the left coronary in dogs. The best results were obtained with arterial autograft, maintaining patency in 7 out of 10 animals.

"Application of the technique to the coronary arteries in human beings was evaluated in autopsy hearts in which an endarterectomy was performed for segmental occlusive disease. The longitudinal arteriotomy incisions in the vessels were closed with a patch thus avoiding narrowing. The patch technique without endarterectomy may be a satisfactory technique for surgical treatment of partial occlusions of the coronary vessels."

*Ellis, P. R., Jr. and Cooley, D. A.: The patch technique as an adjunct to coronary endarterectomy, *J. Thorac. & Cardiovasc. Surg.*, **42:** 236, 1961.

EXPERIMENTAL SURGERY

THE ETIOLOGY AND PATHOGENESIS OF CHOLECYSTITIS: AN EXPERIMENTAL STUDY.*

DONALD J. CURRIE, M.D., F.R.C.S.[C],† *Toronto*

MANY AUTHORS have cast doubt on the importance of bacterial infection in acute cholecystitis. Often, no bacteria are found at the time of operation or, if found, they are the same as those found in cases of chronic cholecystitis. Obstruction of the cystic duct can reasonably be related to the onset of the disease. The pathological changes of hemorrhage and edema, and fewer than the expected number of acute inflammatory cells in the bladder mucosa and wall are generally different from the changes seen in inflammatory lesions in other human organs. Many of these authors favour a chemical causation, such as concentrated bile or activated pancreatic juice. It is accepted that obstruction and stasis of bile are important factors in the pathogenesis of acute cholecystitis.[12]

With the possible exception of the study by Thomas and Womack,[14] cholecystitis has not been produced by bile concentrated by the intact gallbladder. The experiments to be described were designed to investigate the possibility that forced concentration of bile by the gallbladder, a high concentration of cholesterol in the gallbladder or some other substance in human gallbladder bile might produce cholecystitis in experimental animals (Fig. 1).

REVIEW OF THE LITERATURE

In 1938, Aronsohn and Andrews[2] produced acute cholecystitis in dogs by injecting 7 to 20% solutions of certain bile salts or canine gallbladder bile concentrated to about half its volume into the obstructed gallbladder. They were the first to suggest that acute cholecystitis in humans might be produced by bile overly concentrated by the gallbladder. In 1932, Andrews, Schoenheimer and Hrdina[1] showed that the inflamed gallbladder absorbs bile salts rapidly but cholesterol more slowly, if at all. This work has been confirmed by others; it suggests that if overly concentrated bile causes acute cholecystitis, it is due to substances other than bile salts because these salts are rapidly absorbed by the inflamed organ.

In 1942, Womack and Bricker[16] found that acute cholecystitis sometimes occurs after simple ligation of the cystic duct if the bile is left *in situ*. The inflammation varies with the content and concentration of the bile. When the bile is removed and replaced by physiological saline no inflammation occurs.

In 1946, Gatch, Battersby and Wakim[7] injected 5% solutions of commercially prepared bile salts and activated pancreatic juice into the bile ducts of dogs and found that each of these produced acute cholecystitis if the ducts were ligated after the injection. Injection of solutions of bile salts into the portal and systemic veins also produced cholecystitis, but this inflammation was prevented by occlusion of the cystic duct. They concluded that the concentrated bile salts, which produced cholecystitis, reached the gallbladder by way of the common hepatic and cystic ducts.

In 1952, Howard, Milford and DeBakey[8] used a 20% solution of commercially prepared bile salts to produce experimental acute cholecystitis in the obstructed gallbladders of dogs. They found that 15% of the animals had normal gallbladders after this injection, but the percentage of normal gallbladders after injection increased markedly with simultaneous sympathetic denervation.

Thomas and Womack[14] produced a forced concentration of gallbladder bile in

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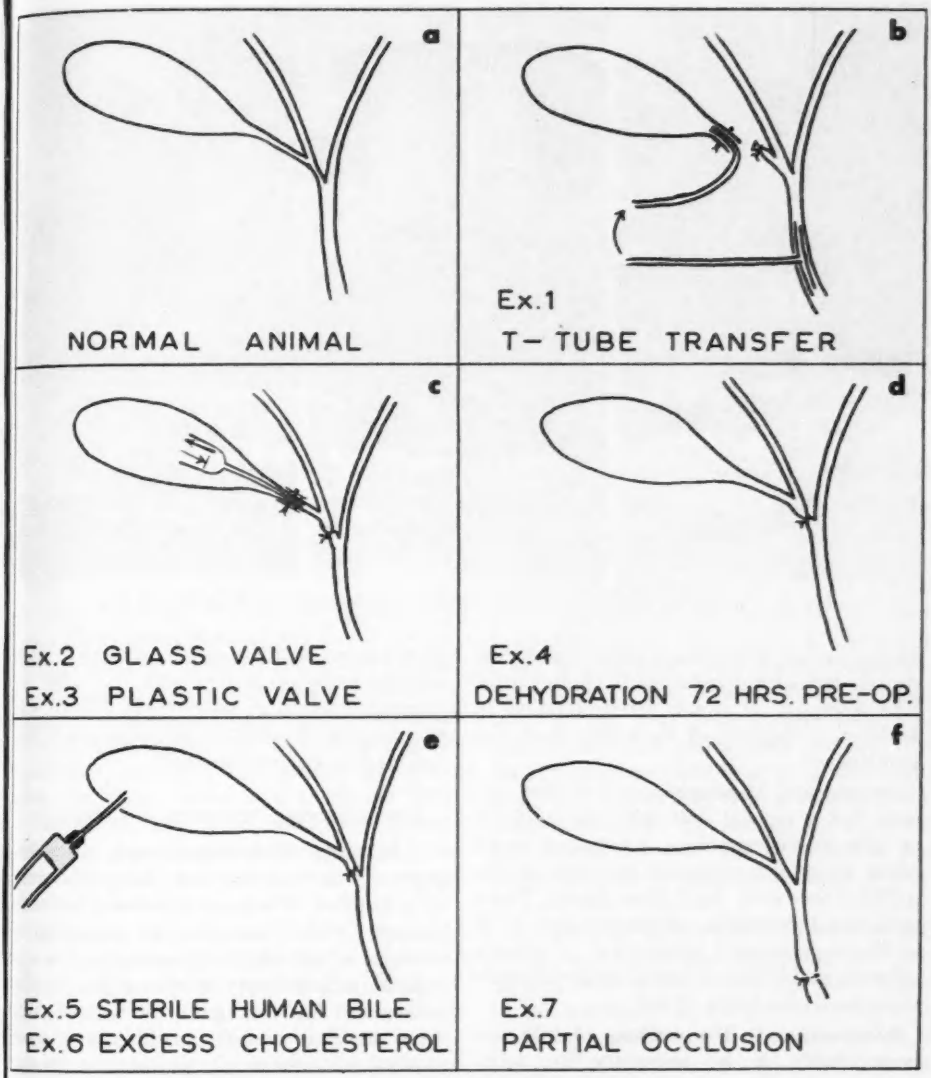


Fig. 1.—An outline of the operations performed in the experiments.

dogs by producing dehydration for 48 hours. After dehydration, ligation of the cystic duct and vessels was carried out and laparotomies were performed at two days, and two, four and eight weeks to study the pathological changes in the gallbladder. Ligation of the cystic duct, cystic artery or separation of the gallbladder from the liver produced no significant change in 10 dogs. Ligation of the cystic duct and artery simultaneously, and replacement of the contained bile with saline produced only slight edema in each of four dogs. Ligation of the cystic duct and artery in four dogs caused total gangrene in three and partial gangrene and edema in the fourth. In 15 dogs, ligation of all of the components of the cystic pedicle *en masse* resulted in six instances of gangrene, six instances of normal gallbladder or mild subacute inflammation, and three instances of a swollen red gallbladder

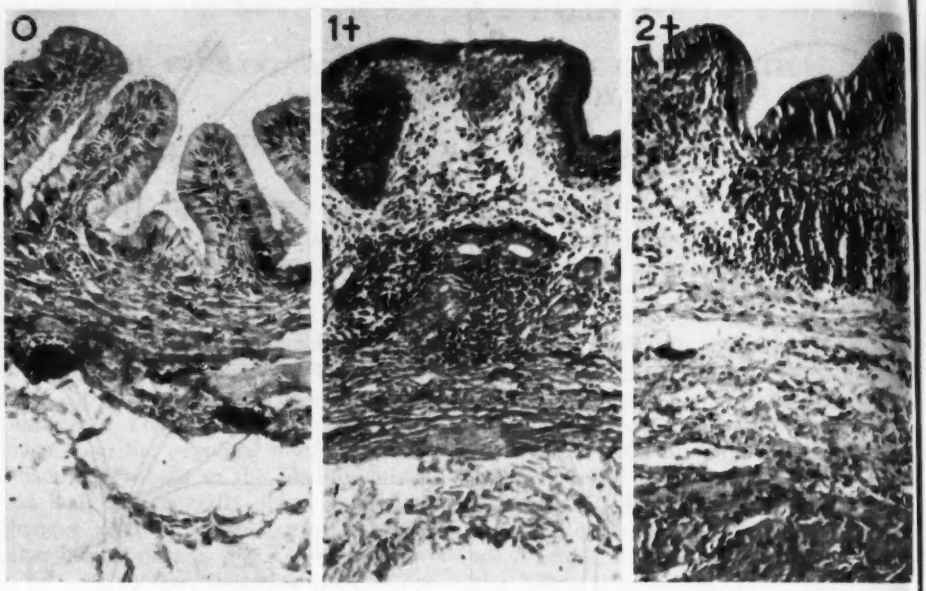


Fig. 2.—The grades of pathological changes found in the control gallbladders of dogs.

bathed in serosanguinous fluid, which was grossly considered to be acute cholecystitis. The necessity of cystic artery ligation in addition to ligation of the cystic duct was emphasized.

Bevans and Mosbach[3] mention that animals fed a normal diet with the addition of dihydrocholesterol or 3-betacholestanol often show inflammatory changes in the gallbladder wall and bile ducts. These cholesterol precursor substances were used in the experimental production of atherosclerosis and biliary calculi. Similar changes were found by Caira *et al.*[5]

Obstruction to the outflow of bile has been shown to be necessary by many workers, including Morris, Hohf and Ivy,[12] in the experimental production of cholecystitis.

In 1957, Imamoglu, Perry and Wangensteen[9] reported the experimental production of biliary concretions by incomplete stricture of the lower common bile duct. Concretions were found in seven of eight rabbits with incomplete stricture after an average interval of nine weeks. There was no mention of inflammatory changes in the gallbladder of these animals.

Experimental Methods and Results

Soon after these experiments were begun, it was realized that the gallbladder of a number of normal laboratory animals showed some microscopic pathological changes which might be mistaken for significant inflammatory reactions due to the procedure. For this reason 64 normal dogs were sacrificed and their gallbladders were studied microscopically as controls for the experiments. Dr. T. Van Patter, pathologist to St. Michael's Hospital, has studied these specimens and all of the specimens from the experiments. He has critically graded each specimen on the basis of inflammatory changes on a basis of zero to four plus. As shown in Table I, the distribution

TABLE I.—The Grades of Inflammatory Change

	Total studied	*0*	*1 plus*	*2 plus*	*3 plus*	*4 plus*
Controls	64	11%	58%	31%	—	—
Experiments	95	8%	48%	29%	10%	5%

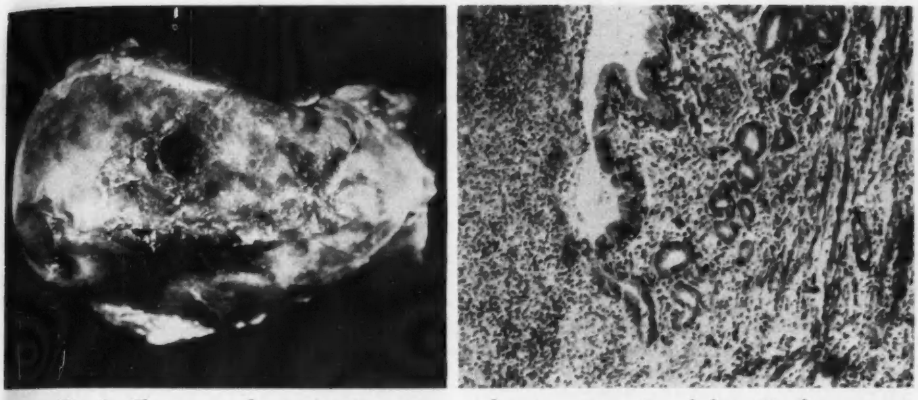


Fig. 3.—The gross and microscopic appearance of acute suppurative cholecystitis from experiment 1. Contamination occurred during repeated transfer of bile to the obstructed gallbladder.

of the grades in the control group closely corresponds to the distribution of the grades in the experimental group. Only grades 3 and 4 plus are considered significant.

Three grades of normal gallbladder from the control group are shown in Fig. 2. On the left, the unquestionable normal is shown. In the middle, there is a slight degree of chronic inflammation with focal lymphocytic infiltration and glandular hyperplasia, and this was graded one plus. On the right, there is a moderate focal lymphocytic infiltration with some muscular hypertrophy graded two plus. All the original photomicrographs were taken at the same power, 13 diameters.

Experiments were designed to force an over-concentration of bile by the gallbladder in dogs by the repeated addition of liver bile, by glass and plastic one-way valves implanted in the cystic duct, by dehydration lasting 72 hours, followed by right hepatic duct ligation, and in rabbits by partial occlusion of the lower common bile duct. Studies of the effect of sterile human gallbladder bile from fresh surgical specimens and of excess commercially prepared cholesterol on the obstructed gallbladder of dogs were also made. All animals had been fed normal kennel rations and fluids *ad libitum* unless otherwise specified. Intravenous pentobarbital was used in every case as the anesthetic agent. Gross colour photographs and histological sections were made of each specimen.

Experiment 1.—T-Tube Transfer of Bile

Repeated injections of liver bile obtained from an indwelling T-tube in the common bile duct were made into the cannulated gallbladder. A No. 10 T-tube was implanted in the common bile duct in 13 dogs in the usual fashion. The cystic duct was isolated by careful dissection and divided. The proximal end was ligated. A No. 240 polyethylene tube was passed into the gallbladder through the distal cystic duct and held by a linen ligature. Bile was collected in a plastic bottle suspended under the animal by a canvas belt. Twice daily, 5-7 ml. of liver bile was transferred to the gallbladder and the plastic tube was clamped.

In spite of very careful dissection and isolation of the cystic duct from the cystic vessels, nine of the 13 animals died or had to be sacrificed in the first five days. In each case, gangrene of the entire wall of the gallbladder was found. The four remaining animals were sacrificed on the second to fourth day after laparotomy; of these, three had necrosis of only part of the gallbladder wall and the viable portions of the wall of these gallbladders showed an intense acute suppurative inflammation indistinguishable from human acute cholecystitis. The other gallbladder had no necrotic areas and showed acute cholecystitis identical to the disease in humans throughout the whole gallbladder wall. The gross and microscopic appearance of this organ is shown (Fig. 3). Cul-

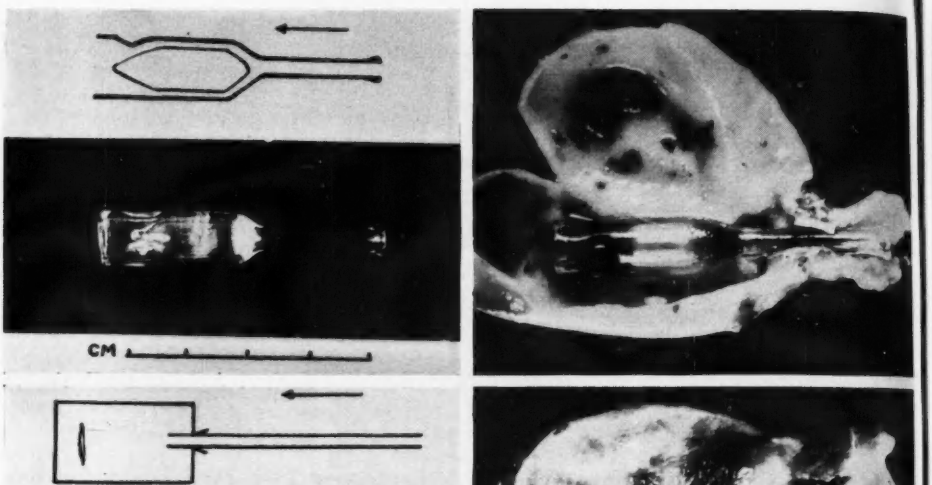


Fig. 4.—The one-way glass and plastic valves used in experiments 2 and 3. On the right, specimens from experiments 2 and 3 are shown in which the action of the glass (above) and plastic (below) valves was fouled by a precipitate of blood, mucus or both.

tures of the contents of all 13 gallbladders were positive for contaminating organisms.

The technical difficulties in this experiment were considerable. In spite of great care in dissection of the cystic duct, gangrene of the gallbladder did occur. We believe that disturbance of the cystic vessels was the cause of gangrene of the gallbladder, although Morris, Hohf and Ivy[12] and Thomas and Womack[14] felt that because of the rich collateral circulation from the liver, simple ligation of the cystic duct or deliberate ligation of the cystic artery could be performed without adverse affect on the gallbladder. Furthermore, it is almost impossible to collect bile continuously from an indwelling T-tube and transfer bile repeatedly to an indwelling plastic tube without contamination. A culture taken from each gallbladder in this experiment showed the presence of contaminating organisms.

Experiment 2.—One-Way Glass Valves

Small one-way valves were made from shaped glass tubing into which a loosely fitting glass obturator was placed to form a tight seal. Each valve was tested before implantation and was found to allow a flow of water in one direction, but did not permit any leakage in the opposite direction. The technical difficulties of fixing the valves in the cystic duct without injury to the vessels were overcome by the use of a small encircling aluminum band which had teeth on its deep surface. The band was held in position around the cystic duct by a ligature. This technique is similar to the method of implanting a Huffnagel valve in the ascending aorta. A short incision in the fundus was made to insert the valve into the lumen of the gallbladder. In most of the animals the right hepatic duct was ligated below the cystic duct to force bile towards the gallbladder.

One-way glass valves were implanted in the cystic ducts of 11 dogs and these animals were sacrificed at varying intervals ranging from 5 to 30 days. The action of all of the valves was fouled by either thick mucus or a precipitate of blood. Microscopical examination revealed a two plus grade of inflammation of a chronic nature in most of the specimens, which was not considered significant. Gangrene did not occur in any of the gallbladders.

Experiment 3.—One-Way Plastic Valves

One-way plastic valves were made from thin polyethylene sheets fused by heat and pressure to form a small flat envelope on the end of No. 240 polyethylene tubing. A small slit was cut in one sheet to allow bile to enter the gallbladder, but lateral pressure on the envelope by bile caused the bag to collapse and effectively prevented bile from leaving the gallbladder. These valves were fixed in the cystic duct by an encircling band, as in experiment 2, and in two of the five animals used the right hepatic duct was ligated below the cystic duct.

As in experiment 2, in every animal the action of the valve was fouled by thick mucus or a mixture of thick mucus and blood (Fig. 4). Microscopically, two specimens showed a three plus degree of subserosal fibrosis and one showed the same degree of mucosal glandular hypertrophy. However, these were found to be isolated changes and each specimen was graded two plus overall, which is an insignificant inflammatory change.

Experiment 4.—Dehydration

Twenty-three dogs were prepared by withholding all fluids for 72 hours before laparotomy. Ligation of the right hepatic duct below the cystic duct was carried out and the animals were sacrificed at intervals ranging from 3 to 87 days. This was an attempt to confirm Thomas and Womack's 48-hour dehydration experiments.[14] Postoperatively, normal food and fluids were provided.

Of the 23 specimens, five showed autolysis from delayed autopsy or improper fixation. One specimen studied on the 64th day showed significant three plus changes reflected in moderate muscular hypertrophy, chronic inflammatory cell infiltration in the mucous membrane and submucosa, and significant lymphoid follicle hyperplasia (Fig. 5). Each of the remaining 17 specimens showed two plus or one plus grades of inflammatory change which were not considered significant.

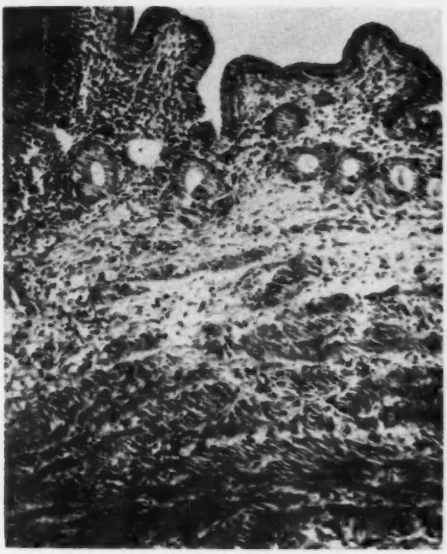


Fig. 5.—A gallbladder from experiment 4 showing significant (3 plus) chronic inflammatory changes.

The technique used differed from Thomas and Womack's experiments in that a 72-hour period of dehydration was used rather than 48 hours, and the right hepatic duct was ligated rather than the cystic duct and vessels *en masse*. Thomas and Womack found three instances of acute inflammation grossly and six instances of gangrene. In our experiment, no instance of gangrene or significant acute inflammation was encountered on microscopic examination. One instance of significant chronic inflammatory change was noted.

Experiment 5.—Sterile Human Gallbladder Bile

In order to investigate the hypothesis that some factor in bile other than microorganisms might be the cause of cholecysti-



Fig. 6.—A gallbladder from experiment 5 showing significant (3 plus) chronic inflammatory changes.

tis, gallbladder bile was collected from human surgical specimens of acute and chronic cholecystitis. The sterility of the bile was confirmed by repeated cultures. After ligation of the right hepatic duct in 15 dogs, the gallbladder was emptied by aspiration and 7 to 12 ml. of the sterile human gallbladder bile was injected into the gallbladder. The hypodermic needle was passed through the liver of the animal directly into the gallbladder to prevent leakage of bile into the peritoneal cavity. Evidence of leakage of bile was not found in any of the animals and studies were made at intervals varying from four to 21 days after laparotomy.

Of the 15 specimens, two were discarded because of autolysis. One specimen studied at 21 days was graded three plus on the basis of moderate glandular hyperplasia, subserosal fibrosis and inflammation with marked chronic inflammatory cell infiltration in the mucous membrane (Fig. 6). The remaining 12 specimens did not have significant inflammatory changes. Sterile human gallbladder bile from cases of acute or chronic cholecystitis did not cause acute cholecystitis in the obstructed gallbladder of the dog in 13 experiments. Only one instance of significant chronic cholecystitis was found.

Experiment 6.—Excess Cholesterol

The effect of an excess of commercially prepared cholesterol on the obstructed gallbladder was observed in 28 dogs. Approximately one gram of sterilized cholesterol crystals was suspended in sterile saline and injected into the emptied gallbladders of the animals after right hepatic duct ligation. No report of a similar experiment was found in the literature. The dogs were sacrificed at intervals ranging from 4 to 138 days.

Of the 28 specimens, eight showed autolysis and in seven of these free bile was found in the peritoneal cavity. Two other dogs died of bacterial peritonitis and another dog was lost. Of the remaining 17 animals, six showed a three or four plus grade of chronic inflammatory change. In these six specimens the changes were described as a moderate infiltration of chronic inflammatory cells in the mucous membrane with marked follicular hyperplasia, moderate muscle hypertrophy and subserosal fibrosis. Clefts found in the thickened subserosa in several specimens were believed to be due to spillage of the suspension of cholesterol crystals into the peritoneal cavity at the time of operation. These six animals were sacrificed 83, 87, 105, 108, 112 and 119 days after laparotomy. Two other animals, sacrificed after 102 and 104 days, were the only other animals studied after more than 65 days, but in these, significant changes were not found. Four of the six specimens with significant pathological changes showed a pebbly granularity of the mucous membrane which was due to marked lymphoid follicle hyperplasia. Three of these gallbladders contained a precipitate consisting of bile salts and blood (Fig. 7). There was no cholesterol in the precipitate. The remaining 11 specimens did not show significant pathological changes and nine of these animals were sacrificed before the 65th postoperative day. Prolonged exposure of the mucous membrane of the obstructed gallbladder to excess cholesterol may be a

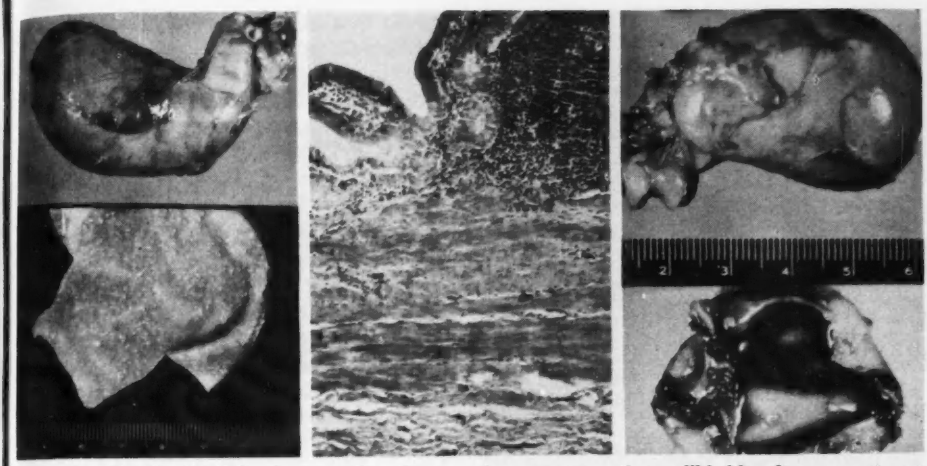


Fig. 7.—On the left, the serosal and mucosal appearance of a gallbladder from experiment 6 showing significant inflammatory changes on the 105th day. Note the pebbly granularity of the mucous membrane caused by lymphoid follicle hyperplasia. In the middle, the microscopic appearance of the chronic inflammatory changes graded 4 plus. On the right, the intact and opened appearance of another gallbladder from experiment 6 is shown. In addition to significant (3 plus) chronic inflammatory change this specimen had a precipitate of bile salts on the 108th day.

cause of chronic cholecystitis. No evidence of acute inflammatory change was found.

Experiment 7.—Partial Occlusion of the Lower Common Bile Duct in Rabbits

An experiment similar to that reported by Imamoglu, Perry and Wangensteen[9] was performed to investigate the hypothesis that incomplete stricture of the terminal common bile duct in rabbits might cause significant inflammatory changes in the gallbladder or bile ducts. Instead of a wrapping of cellophane sealing tape dusted with dicetyl sodium phosphate powder as used by Imamoglu, Perry and Wangensteen, a ligature of coarse string was tied around the lower common bile duct of 45 rabbits using two strands of 0 chromic catgut as a spacer. The in-

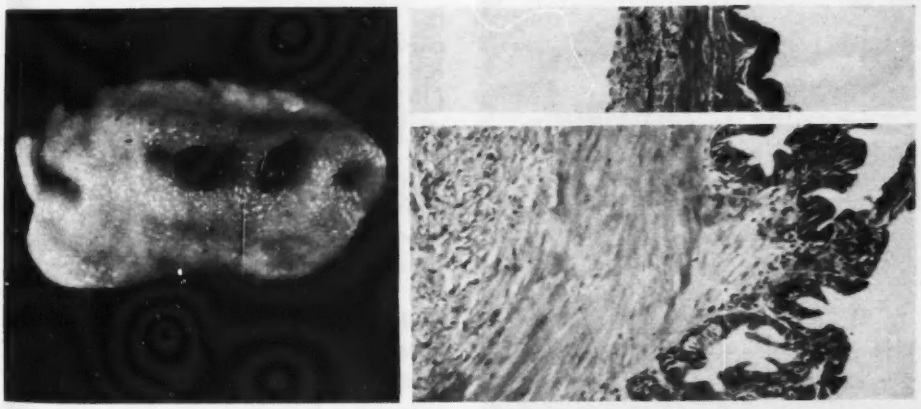


Fig. 8.—The gallbladder of a rabbit from experiment 7 after 175 days. Two large calculi are shown on the left in the opened specimen. Note the roughened mucous membrane. In the upper right, the appearance of a normal rabbit's gallbladder is shown for comparison. In the lower right is shown the significant (4 plus) chronic inflammatory change from the gallbladder on the left. Marked glandular hyperplasia, muscular hypertrophy and subserosal fibrosis are seen.

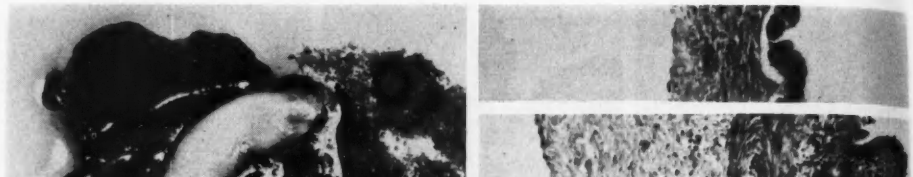


Fig. 9.—The gallbladder of another rabbit from experiment 7 studied on the 152nd day. The heavy precipitate of small particles consisting of bile salts and blood is shown. A photomicrograph of a normal rabbit's gallbladder is shown in the upper right for comparison. The significant (3 plus) chronic inflammatory changes in this animal's gallbladder are shown in the lower right.

tervals between operation and autopsy varied from 2 to 175 days.

No evidence of significant acute cholecystitis was found in any of the 45 specimens. Eighteen rabbits died unexpectedly and these specimens were unsuitable for careful study because of autolysis. Of the remaining 27 rabbits, 21 were studied within 40 days after laparotomy and although some specimens showed some postmortem change, there was no instance of significant acute or chronic inflammatory change when compared with a number of normal rabbits' gallbladders which were used as controls. Specimens of the six rabbits studied more than 40 days after laparotomy all showed significant chronic inflammatory changes. There was moderate to marked muscular hypertrophy and subserosal fibrosis. The mucous membrane showed moderate glandular hypertrophy and chronic inflammatory cell infiltration with thickening and round cell infiltration in the submucosal layer. The gallbladder walls were at least five times thicker than the controls after making allowance for uneven shrinking during fixation. The three rabbits which survived for the longest time (152, 162 and 173 days) had biliary concretions in the gallbladder and bile ducts. The calculi were firm but not hard and consisted of bile salts with varying amounts of blood pigment. The precipitates contained very little cholesterol. Five of the six animals which survived for long periods had obvious biliary cirrhosis (Fig. 10). Each animal had an incomplete occlusion when tested at the time of autopsy. These animals were not jaundiced (Figs. 8-11).



Fig. 10.—Photomicrograph of the periductal inflammation and fibrosis in the liver of the rabbit studied on the 152nd day.

Discussion

Acute cholecystitis in the human usually develops in a gallbladder which has evi-

dence of chronic cholecystitis and cholelithiasis. There is generally an obstruction of the cystic duct, usually by a gallstone. In rare cases where a gallstone is not found, the obstruction may have been transient during the development of the inflammation.

Infection.—The microscopic changes of human acute cholecystitis are unusual in that there is gross edema and hemorrhage and fewer than the expected numbers of acute inflammatory cells when compared with acute inflammatory lesions in other human organs. This unusual microscopic picture does not indicate necessarily that acute cholecystitis is a non-infectious disease and should not be accepted as proof that there is a chemical causation. It can be argued that this peculiar appearance is due to the presence of the pre-existing changes of chronic cholecystitis complicated by acute cystic duct obstruction. In experiment 1, bacterial infection was found in every instance of acute cholecystitis. Acute cholecystitis was not found in any of the other experiments and all other cultures were negative. Bacterial infection may still be an important factor in the pathogenesis of human acute cholecystitis. The infection is not specific; the same organisms are often cultured from specimens of chronic cholecystitis. The important additional factor in acute cholecystitis is stasis caused by an acute obstruction to the outflow of bile from the gallbladder. It is likely that the relative importance of obstruction and infection varies in individual instances and this may account for the variation in the pathological changes. In these experiments, acute cholecystitis was not found as a complication of chronic cholecystitis in any of the animals that had significant chronic inflammatory changes.

Chemical Agents.—There is little doubt that artificially concentrated bile, 5 to 20% solutions of commercially prepared bile salts and also activated pancreatic juice can cause acute cholecystitis in the obstructed gallbladder of the experimental animal.[2, 4, 7, 8, 16] The results of experiments 2, 3, 4, 5 and 7, in which 47 dogs and 27 rabbits were studied, failed to demonstrate that the gallbladder of the dog or rabbit can concentrate bile sufficiently to produce acute cholecystitis. Thomas and Womack[14] state that 48 hours of dehydration did not result in a concentration of bile by the gallbladder as great as that used by others to produce experimental acute cholecystitis. It has never been shown that the gallbladder is capable of concentrating bile to the degree required to produce acute cholecystitis. Animals in these experiments were studied at varying intervals following laparotomy and it is unlikely that phases of acute inflammation were missed. Acute cholecystitis in humans usually occurs in gallbladders which have the changes of chronic cholecystitis, and it can be shown radiographically that most of these gallbladders are incapable of concentrating bile.



Fig. 11.—The gross appearance of liver, gallbladder and precipitate from the rabbit in experiment 7, studied on the 162nd day after laparotomy. The gross changes of biliary cirrhosis are seen, and in this gallbladder the concretions were dark, due to a higher proportion of blood pigment in the bile salt-blood pigment precipitate. The gallbladder wall had a 4 plus degree of chronic inflammatory reaction.

Chronic Cholecystitis.—Interesting observations were made concerning the development of significant chronic cholecystitis in these experiments. Prolonged stasis caused by obstruction to the outflow of gallbladder bile is an important factor. One specimen, of the 18 studied in experiment 4, in which the right hepatic duct was ligated after 72 hours of dehydration showed significant chronic inflammatory changes after 63 days. In experiment 5, in which sterile bile from cases of human cholecystitis was injected into the gallbladder after right hepatic duct ligation, one of the 13 specimens studied showed significant chronic inflammation after 21 days. In experiment 6, in which excess sterile commercial cholesterol was injected into the obstructed gallbladder, six of eight specimens studied after 82 days had significant chronic cholecystitis. In experiment 7, in which the lower common bile duct was partially occluded, the gallbladders of all six rabbits that were studied after 40 days had significant chronic inflammatory changes.

Cholesterol.—Rabbit's bile has a higher concentration of cholesterol than dog's bile and it may happen that a relatively high concentration of cholesterol in bile is a significant factor in the development of chronic cholecystitis. This is suggested by the results of experiment 7 using the rabbit, experiment 6 where excess cholesterol was used and experiment 5 in which sterile human gallbladder bile was injected into the obstructed gallbladders of dogs. In humans, a high cholesterol concentration in bile in diabetes mellitus, obesity, during pregnancy and in other conditions is associated with a higher incidence of chronic cholecystitis and cholelithiasis.[16]

Calculi.—Calculi were found in the gallbladders of three dogs in experiment 6 and of three rabbits in experiment 7. These stones consisted mainly of bile salts, a variable amount of blood pigment but little cholesterol. This is the composition of biliary concretions produced experimentally by others.[5, 9] All of these animals were studied after a considerable period of time following laparotomy. No mucosal deposit of cholesterol was found. Canine bile contains less than one-fifth the concentration of cholesterol as compared with human gallbladder bile and human cholesterol gallstones rapidly dissolve in dog's bile.[6] Cholesterol is very slowly absorbed by the gallbladder, but the absorption of bile salts by the gallbladder and the excretion of bile salts by the liver created a local circulation of bile salts because of the right or common bile duct ligation in these experiments. This circulation apparently greatly assisted the absorption of cholesterol by the gallbladder. Apparently the relatively low concentration of cholesterol in dog's and rabbit's bile prevented the precipitation of cholesterol in experiments 6 and 7.

In the human it is possible that a high concentration of cholesterol in the bile, perhaps due to some metabolic dysfunction,[6, 15] is the primary predisposing condition in these diseases of the extrahepatic biliary system. The cholesterol may precipitate to form calculi and either the excess cholesterol or the calculi initiate the changes of chronic cholecystitis. Gallstones are found in human gallbladders which otherwise could be considered normal. Acute cholecystitis then might occur because of acute cystic duct obstruction. Perhaps infection plays a part in the development of acute cholecystitis. The concentration of cholesterol in the bile is increased by stasis. The stasis is due to incomplete obstruction to the outflow of bile from the extrahepatic biliary tree but the nature of the partial obstruction is not clear. As Wangensteen and others have described, almost invariably we have found some resistance to the passage of a 3 mm. dilator into the duodenum during common duct exploration in cases of chronic or acute cholecystitis, independent of other positive findings in the duct. Anatomical or functional variations such as tortuosity, villous projections and spasm have been suggested as the cause of the narrowing at the lower end of the common bile duct. Stasis resulting in an increased concentration of bile in the human is aggravated by paralytic ileus, dehydration and morphine administration and is believed to be very

important in the development of postoperative cholecystitis, recurrent stones and cholesterol sludge in the lower common bile duct.

Summary and Conclusions

Very rigid criteria for determining significant degrees of inflammation are required in the interpretation of the results of experiments on the gallbladders of animals, particularly the dog.

Acute cholecystitis developed only in the presence of bacterial infection and obstruction to the outflow of bile from the gallbladder. It did not occur in the absence of infection. Acute cholecystitis could not be produced by a forced over-concentration of bile by the intact gallbladder brought about by such measures as prolonged stasis by duct ligation, one-way valves and dehydration in 47 dogs and 27 rabbits. The likelihood that acute cholecystitis is a complication of chronic cholecystitis, acute cystic duct obstruction and possibly infection, was discussed.

Chronic cholecystitis was produced by prolonged stasis due to partial obstruction to the outflow of bile in six rabbits and in six dogs where excess cholesterol was added to the bile. The gallbladders of three of these rabbits and three of these dogs contained biliary precipitates after 40 days. In agreement with the findings of others,[5, 9] the experimental biliary concretions consisted mainly of bile salts and little cholesterol. The high cholesterol content of human bile and factors which increase the concentration of bile were discussed in relation to the pathogenesis of cholelithiasis and chronic cholecystitis.

The author wishes to express his deep appreciation to Dr. T. Van Patter for reviewing and critically grading all specimens and to Mr. Arthur Smialowski for the preparation of prints from 35 mm. original colour transparencies and, in cooperation with Dr. Van Patter, for the photomicrographs. He would also like to thank Mr. J. A. Durno of Canadian Industries Limited for his kindness in making the plastic valves. Professors W. Keith Welsh and F. G. Kergin have kindly read and criticized the manuscript.

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Résumé

Cet article rapporte les résultats obtenus par l'auteur dans une étude de pathologie expérimentale. Dans ce type de recherches, il est nécessaire d'établir des critères très rigides quant à l'estimation des résultats, particulièrement chez le chien.

Une vraie cholécystite aiguë ne peut se développer qu'en présence d'une infection bactérienne et d'une obstruction des voies biliaires provoquant une stase dans la vésicule. L'obstruction seule est insuffisante. Une sur-concentration de bile dans une vésicule intacte, obtenue par la

ligature des canaux, un système de valvules à sens unique ou la déshydratation ne donna aucun effet chez 47 chiens et 27 lapins.

L'auteur discute ensuite les possibilités existantes que la cholécystite aiguë soit la suite de la cholécystite chronique lors d'une obstruction subite des canaux. La cholécystite chronique a pu être provoquée chez six lapins et six chiens par la stase intra-vésiculaire; celle-ci fut obtenue par une ligature partielle des canaux et une surcharge en cholestérol. Après une quarantaine de jours, on trouva des précipités biliaires chez trois de ces lapins et trois des chiens. Ces précipitations étaient formées principalement de sels biliaires et d'un peu de cholestérol. Les relations possibles entre ces expériences et ce qui est rencontré chez l'homme sont exposées et discutées.

SOME OBSERVATIONS ON PERIPHERAL BLOOD FLOW, BLOOD GAS, AND ELECTROLYTE CONTENT OF THE DOG'S LIMB AFTER SYMPATHECTOMY*

R. K. PADHI, F.R.C.S.[C]† and R. B. LYNN, F.R.C.S.,† *Kingston, Ont.*

THE HYPEREMIA which occurs after sympathetic denervation gradually subsides so that the initial profound effect of the operation is not maintained. It has been shown that after sympathectomy the residual flow in the foot is still double the initial flow after two to three months, whereas in the hand the flow returns to almost the original level within two weeks.[1] The question still remains, what brings about the return of vascular tone? Burn[2] has suggested that the return of vascular tone is due to enzymatic factors since it runs a course parallel with the level of amine oxidase. However, there is no unanimous agreement and the mechanism of the return of vascular tone still remains obscure.

The oxygen saturation of the venous blood in a sympathectomized limb is markedly increased and this is believed to be due, in part, to the opening up of the arteriovenous shunts. If this hypothesis is correct, the oxygen saturation of the venous blood in a sympathectomized limb should diminish as vascular tone returns, unless the failure of blood flow to return to its initial level is the result of some residual paralysis of these arteriovenous (A-V) shunts. This relationship, however, has not yet been established but is suggested by the persistent elevation of skin temperature after sympathectomy.[1]

Secondly, Lyman[5] and others have suggested that sympathectomy causes increased permeability of the cell membrane to potassium; this results in the migration of intracellular potassium. On the other hand, Beaconsfield‡ reported a transient fall in serum potassium immediately after sympathectomy.

The purposes of this study have been first, to evaluate the relationship, if any, between the return of vascular tone and oxygen saturation of the venous blood in a sympathectomized limb; secondly, to determine any alterations in the electrolyte content of the venous blood leaving a sympathectomized limb.

MATERIAL AND METHODS

Twenty unselected mongrel dogs, weighing between 8 and 10 kg., were divided into three groups:—

Group I.—Ten dogs were anesthetized with sodium pentobarbital. The volume of blood flow in the hind paws was determined and blood samples were obtained from both hind paws for blood gas and

*Presented at the 1961 Annual Meeting of the Royal College of Physicians and Surgeons of Canada, Ottawa, Ont.

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‡Beaconsfield, P.: Ph.D. thesis, University of London (personal communication).

electrolyte determinations. The right lumbar sympathetic chain was exposed retroperitoneally through a muscle-splitting incision and all the ganglia encountered between the crus of the diaphragm and the brim of the pelvis were removed and the wound was closed in layers. One hour after sympathectomy, the volume of blood flow in the limbs was again measured and blood samples were obtained for blood gas and electrolytes. Further studies were done at regular intervals up to one month postoperatively.

Group II.—Five dogs were lightly anesthetized with sodium pentobarbital. The right common femoral artery was exposed and ligated in four places, and divided to render the right hind paw ischemic. Five to six weeks later these animals were again anesthetized with sodium pentobarbital. Blood samples were obtained from both hind paws for blood gas and electrolyte determinations. After recording the volume of blood flow in the hind paws, right lumbar sympathectomy was carried out. One hour after sympathectomy blood flow volumes were again measured and blood samples obtained for blood gas and electrolyte determinations. Further studies were done at regular intervals up to one month postoperatively.

Group III.—Control Group—Five animals were anesthetized, blood flow measurements in the hind paws were taken and blood samples from both hind paws were obtained for blood gas and electrolyte determinations. The right sympathetic chain was exposed in the same way as in Groups I and II and the wound was closed in layers without removing the ganglia. Although the sympathetic chain was exposed by retroperitoneal dissection, the chain was not intentionally traumatized. The volume of blood flow was recorded and blood samples were obtained an hour after this procedure for blood gas and electrolyte determinations.

Blood flow was measured by venous occlusion plethysmography. A plethysmograph was attached to each hind paw and rendered air-tight with strip-seal putty. The measurements were recorded on a Grass plethysmograph.

Oxygen saturation was determined by using the Beckman DU spectrophotometer,[3] the carbon dioxide by the Van Slyke method, and the serum sodium and potassium by using the Unicam flame spectrophotometer (SP 900). Serum chloride was measured by the electrometric method described by Lehmann.[4]

Follow-up

All the animals were followed up for over 30 days. Blood flow, blood gas and electrolyte determinations were carried out on the first, second, third, fifth, seventh, tenth, fifteenth, twentieth and thirtieth days.

Results

Blood Gases

In the control group (mock sympathectomy) the average oxygen saturation of the venous blood in the paw rose from 57% to 72% within an hour. Within 48 hours the saturation had returned to the previous level and showed no further alteration (Fig. 1).

In Group I (the animals with normal vasculature) the oxygen saturation rose from the initial level of 57% to 89% within an hour in the sympathectomized limb (Fig. 2). The oxygen saturation remained near this level for approximately three days

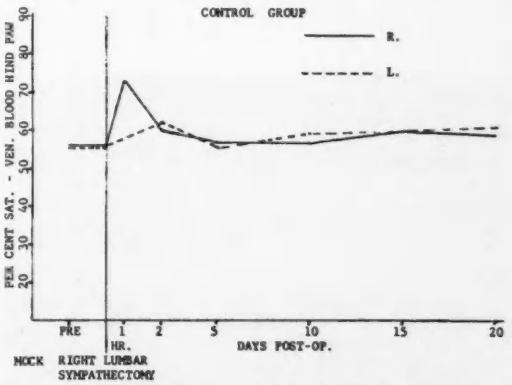


Fig. 1.—A comparison of the oxygen saturation of the peripheral venous blood in the right and left hind limbs in dogs that had a mock operation.

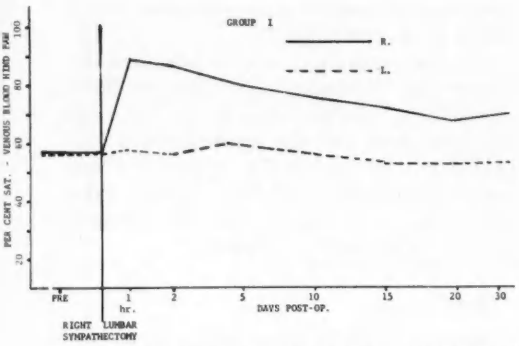


Fig. 2.—The persistent elevation of peripheral oxygen saturation in the sympathectomized limb.

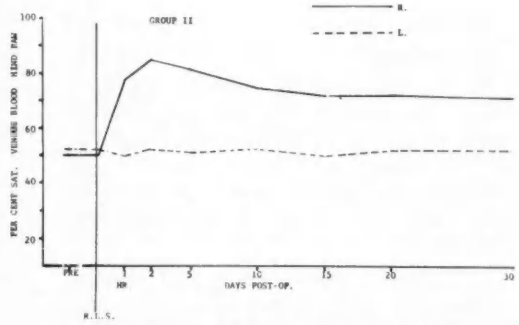


Fig. 3.—A persistent elevation of the oxygen saturation after sympathectomy. In the ischemic right limb the rise to the peak saturation is delayed.

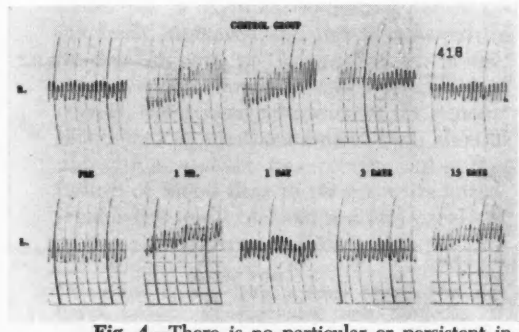


Fig. 4.—There is no particular or persistent increase in peripheral blood flow following a mock operation.

and from then on slowly fell, reaching a plateau about the tenth day. On the thirtieth day, the difference in the oxygen saturation of the venous blood in the two hind paws was still 18%.

In Group II (right hind limb rendered ischemic) the rise in oxygen saturation after sympathectomy was delayed. Oxygen saturation of the venous blood of a sympathectomized limb had risen from 50% to 78% within an hour (Fig. 3). On the second day after sympathectomy, the oxygen saturation had further increased to 85%. From the third day on, the oxygen saturation gradually diminished reaching a plateau on the tenth day. On the thirtieth day, the oxygen saturation of the venous blood in the sympathectomized limb was 72%, 16% to 18% higher than in the normal limb.

The carbon dioxide content of the venous blood in the two hind paws was measured in all the animals. There were no significant alterations in the CO_2 content of the venous blood in any of the three groups studied.

Blood Flow

In the control group there was a transient rise in blood flow. On the second day, however, the hyperemia subsided and no further alterations were observed in the blood flow (Fig. 4).

In Group I (with normal vessels) there was an immediate increase in the blood flow to 300% of normal (Fig. 5). From the third day after sympathectomy, the flow gradually diminished reaching a plateau about the tenth day. At one month the blood flow was still double the initial value.

In Group II (with arterial obstruction) increase in blood flow was more gradual. At the end of one hour the blood flow had increased from 2.5 mm. to 12.5 mm. and 24 hours later it had further increased to 25 mm., that is ten times greater than the initial flow (Fig. 6). From the third day on, the blood flow gradually diminished reaching a base line about the sixteenth day. At the end of the month, the blood flow was still substantially higher than the initial level.

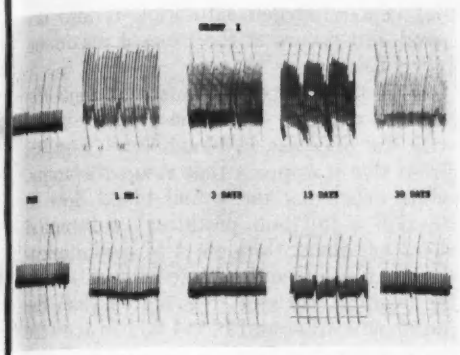


Fig. 5.—The persistent increase in peripheral blood flow following sympathectomy.

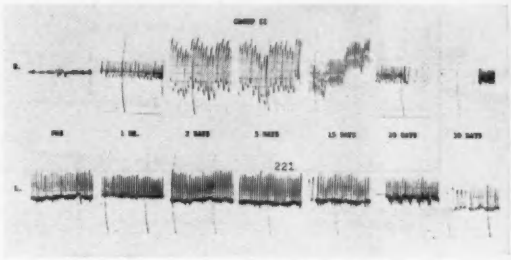


Fig. 6.—With arterial obstruction the increase in flow is delayed but is persistently elevated up to 30 days.

Serum Electrolytes

Potassium, sodium and chlorides were the three electrolytes measured in this study.

Potassium.—In Group I there was a transient fall in the level of serum potassium in the sympathectomized limb. On the second day, however, the level of potassium in both limbs had reached the same level. In Group II the fall of potassium at the end of one hour after sympathectomy was seen in both limbs (Fig. 7). Again, in the control group where sympathectomy was not done, the serum potassium showed a gradual but transient fall. Thus, a transient fall of potassium in Group I and Group II appears to be of no significance.

Sodium.—(Fig. 8). In all three groups the serum sodium level showed no significant alteration.

Chlorides.—These similarly showed no alteration in any of the three groups.

Discussion

It is of interest that in the control group where the sympathetic chain was merely exposed and not removed, immediate vasodilatation occurred. This hyperemia was transient however. This may explain the temporary beneficial effects sometimes seen clinically after inadequate sympathectomy or failure to remove any of the ganglia.

Maximum vasodilatation occurred immediately after sympathectomy in the presence of normal vessels. Maximal increase in oxygen saturation also occurred immediately, whereas with the major artery obstructed, the maximal change was delayed by about 24 hours. These findings agree with blood flow studies done on the human subject. Sympathetic denervation by releasing vascular tone would be expected to produce immediate vasodilatation and this is what happens in the presence of non-obstructed vessels. The delay in the

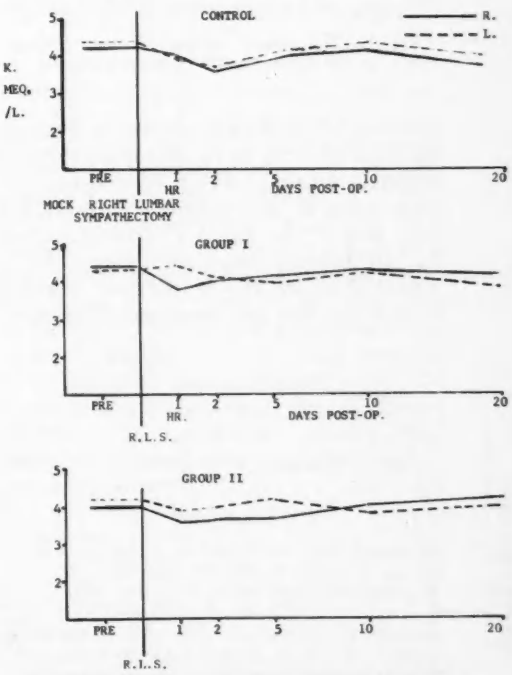


Fig. 7.—The changes in serum potassium with the mock operation, with sympathectomy in normally vascularized limb, and in sympathectomy with ischemic limb.

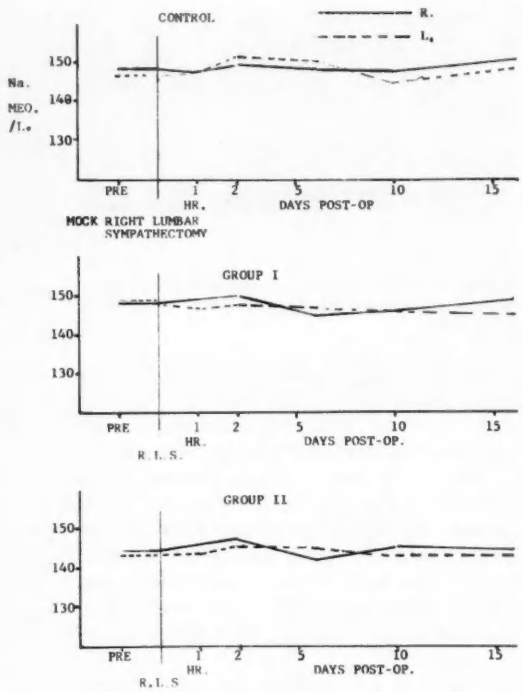


Fig. 8.—The serum sodium changes that occurred in the three groups of dogs studied.

presence of obstructed arteries is because the limb receives its blood supply through collateral channels which do not immediately dilate to their maximal capacity but relax slowly. Too, the A-V communications do not become maximally patent for a period of hours; this is probably reflected by the fact that the development of maximal venous oxygen saturation is also delayed and occurs at the time of maximum blood flow.

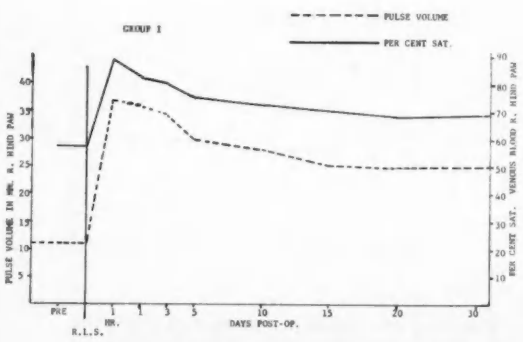


Fig. 9.—The persistent elevation in blood flow and blood saturation following sympathectomy.

With the recovery of vascular tone the venous oxygen saturation falls parallel with the decrease in blood flow (Fig. 9). From this it appears that sympathectomy, while improving the actual blood flow to the skin of the limb, produces a substantial shunt of blood through A-V communications. After a month, oxygen saturation on the average was still 16% higher than the initial saturation and blood flow was twice the preoperative level.

These experimental observations were confirmed clinically when a 48-year-old carpenter was investigated for ischemic changes in the right middle and ring fingers. The clinical impression of Raynaud's disease was confirmed by arteriography. Blood flow determination, oxygen saturation of the venous blood and serum electrolyte content in both hands were recorded before and after cervicodorsal sympathectomy (Fig. 10). The findings were similar to those seen in the experimental animal. Here again, no alterations in the electrolyte content were noted.

Partial recovery of vascular tone starts on the third day and is well established by the fifth day. What brings back the vascular tone? Several theories have been advanced. Burn has suggested that the return is due to enzymatic changes because the level of amine oxidase falls after sympathectomy and is later partially restored. Incomplete denervation and hypersensitivity of the denervated blood vessels have also been suggested as causes of the return of vasomotor control, but neither are felt to be active here because blood flows were still elevated 30 days after denervation. If the former, incomplete denervation, were important, the return of vasomotor tone would have been more rapid and complete.[7] It is most probable that the return of vasomotor tone is a result of the recovery and/or development of intrinsic vascular tone, and this is supported by the finding that intra-arterial tolazine hydrochloride (Priscoline) causes intense vasodilatation in the completely denervated limb.[8]

The present study does not throw any

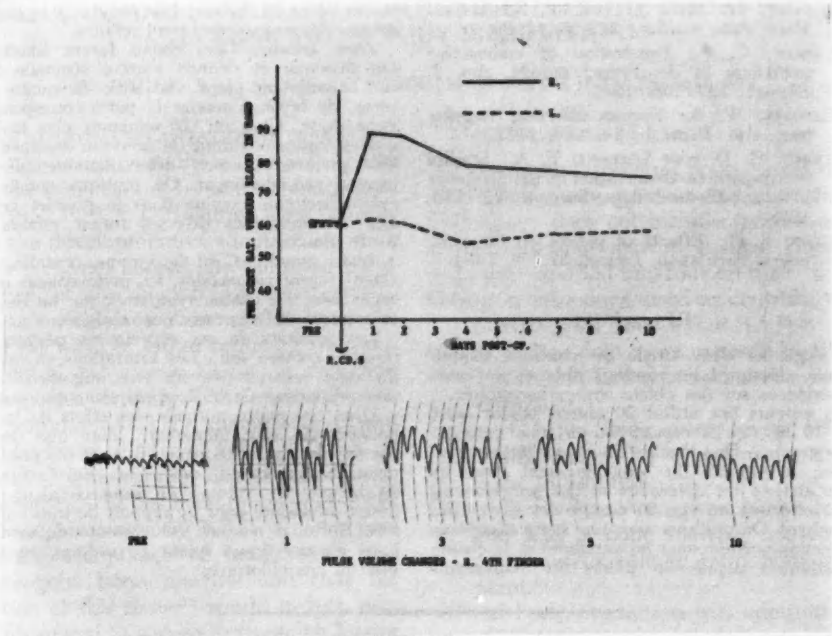


Fig. 10.—The elevation of oxygen saturation in the peripheral blood of the hand and the elevation in the peripheral blood flow following cervicodorsal sympathectomy in a human subject.

light on the mechanism of the return of vasomotor tone in the limb after sympathectomy. It does suggest that there is some degree of permanent paralysis of A-V anastomoses because it is not felt that the persistent elevation of venous oxygen content could be the result of the small persistent elevation of blood flow alone. This concept is supported by the fact that peripheral skin temperatures remain permanently elevated after sympathectomy even though the blood flows have subsided considerably.[1] It is also felt that sympathetic denervation does not cause any measurable alteration in the transfer of electrolytes across the cell membranes as has been suggested by Beaconsfield and another author.[5]

Summary

The effects of sympathectomy on blood flow, oxygen saturation of the venous blood and electrolyte content of the dog's paw have been studied. Oxygen saturations of the venous blood of a sympathectomized limb increases up to 88% and with the return of vascular tone the saturation gradually falls. In a limb with normal arteries the maximum effect of sympathectomy is immediate, whereas in a limb with abnormal arteries the maximum vasodilatation occurs in about 24 hours. A month after sympathectomy the blood flow was found to be three to five times the original level. The oxygen saturation of the venous blood and blood flow in a sympathectomized limb fall consonantly with the return of vascular tone. No significant alteration in potassium, sodium and chlorides occurred after sympathectomy.

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Résumé

Il s'agit ici d'un article de chirurgie expérimentale, décrivant les résultats obtenus au cours d'expériences sur des chiens sympathectomisés.

Les auteurs ont utilisé 20 chiens pesant entre 8 et 10 kg., qu'ils répartirent en trois groupes:

1er groupe.—Une dizaine de chiens furent anesthésiés; le volume du sang circulant dans les pattes arrières fut déterminé et des prélèvements furent effectués en vue du dosage des gaz et des électrolytes. On pratiqua alors une sympathectomie très étendue comprenant les ganglions et la chaîne sympathiques depuis les piliers du diaphragme jusque dans le bassin. Une heure plus tard les mêmes déterminations furent refaites.

2ème groupe.—Cinq chiens furent anesthésiés. On disséqua et dénuda l'artère fémorale droite sur laquelle on plaça une série de quatre ligatures, de façon à rendre la patte correspondante ischémique. Cinq ou six semaines plus tard, les mêmes animaux furent de nouveau anesthésiés et des prélèvements et déterminations effectués comme précédemment. On pratiqua ensuite une sympathectomie comme dans le premier groupe. Les déterminations diverses furent refaites une heure plus tard.

3ème groupe.—C'est le groupe contrôle. Cinq chiens furent anesthésiés; les prélèvements et dosages faits. La chaîne sympathetique fut dénudée et exposée à l'air, mais non réséquée.

Les résultats de ces expériences peuvent être résumés comme suit: Les saturations en oxygène du sang veineux peuvent être augmentées dans une proportion de 88% après sympathectomie.

Dans une patte normale, les effets de la sympathectomie sont immédiats, alors que dans le cas des pattes où les artères avaient été préalablement ligaturées, il existe un délai d'environ 24 heures. Le volume du sang circulant a été trouvé augmenté dans un rapport de trois à quatre fois. Enfin, il n'a pas été constaté de modifications électrolytiques quant au sodium, au potassium ni aux chlorures.

PROSTHETIC VALVES FOR CARDIAC SURGERY. Editor-in-Chief, K. Alvin Merendino, M.D., Department of Surgery, University of Washington, School of Medicine. 586 pp. Illust. Charles C Thomas, Springfield, Ill., 1961. $8.25.

This publication is the accumulated papers presented at the International Symposium on Prosthetics for heart surgery held in Chicago, September 9 and 10, 1960. At this meeting almost all the workers in the Western world interested in the various aspects of heart valve replacement were present.

During the two-day study nearly all aspects of problems related to heart valve replacement were reviewed and these have been put together in this bound volume under the editorship of Dr. Merendino of Seattle. For anyone interested in the problems of mitral or aortic valve surgery this is an invaluable report. The first portion of the papers presented deals with the mechanics of heart valve action and myocardium metabolism. Not only are the papers of extreme importance but also a tremendous amount of knowledge was imparted in the ensuing discussions which are included in the publication. This again brings out the point that often the discussion created by scientific papers is as interesting or more interesting than the actual presentation. Certainly at this conference it was important.

The second series of papers were on all the various plastics and materials that are used for heart valve replacement. The basic problem of thrombosis occurring in animals, when portions of their mitral or aortic valves were replaced, played a prominent role in the presentation. The general differences between replacement in dogs and replacement in humans, as far as animal differences are concerned, is discussed in great detail.

One of the most exciting results that came out of efforts at replacement of the mitral valve was the success by the group at the National Institute of Health led by Dr. Braunwald and Dr. Morrow in totally replacing a human mitral valve. In their report they described the method of construction and insertion of the valve; unfortunately the patient had died at the time of presentation but had lived for a period of three months with a totally replaced mitral valve. Dr. Starr reported extremely encouraging results in the use of a ball-valve prosthesis for the mitral valve and since has had five patients survive after total mitral valve replacement.

As far as aortic valve replacement is concerned, the study by Dr. Hufnagel in Washington of 100 patients subjected to leaflet subcoronary replacement was a most encouraging paper. He had followed up some of these patients for as long as one year and they had done extremely well.

As a result of this conference it became evident that replacement of the aortic valve was an established procedure. Replacement of the mitral valve presents more complex problems.

In summary this is an extremely exciting volume for any surgeon working in open heart surgery with acquired heart disease.

SPLENIC AND BONE MARROW HOMOGRAFTS IN THE DOG AFTER LETHAL TOTAL BODY IRRADIATION*

J. W. IRVINE, B.Sc., M.D. and S. KLING, M.D., F.R.C.S.[C], *Edmonton, Alta.*

WHILE AUTOTRANSPLANTATION of organs has been almost universally successful in the past, the same cannot be said of homotransplantation.[14, 15, 19] Difficulties seem to stem from the development of the immune state by the recipient animal and the subsequent rejection of the homotransplanted organ. Attacks on this rejection mechanism have taken the form of either reducing the antigenicity of the donor tissue, or reducing the response of the host. Adrenocorticotrophic hormone (ACTH), splenectomy,[20] and total body irradiation (T.B.R.), have all been tried with varying degrees of success. In rodents[2, 3, 9] it has been found that lethal total body irradiation (T.B.R.) makes possible the acceptance of homologous and heterologous bone marrow and that the injection of this tissue[11] would in turn permit the animal to survive for a much longer period of time. After one to two months the animal becomes ill again and a phase termed "secondary disease" is encountered. This is believed to be a reaction between the graft and the host which is brought about by the return of the animal's own bone marrow function.[12] If the animal survives this crisis the subsequent course is free from such serious reactions. In dogs[5] the injection of bone marrow alone appears to give inadequate protection against the effects of T.B.R. inasmuch as the animal's immune mechanisms are still crippled. The formed elements of the blood return in adequate numbers with the exception of lymphocytes.

Our present experiment was designed to remedy this deficiency by the addition of spleen as a source of lymphocytes. It was realized that this step might entail further risk as well as potential benefit; for although the addition of spleen, the largest single reservoir of lymphocytes, might permit the animal to be biologically self-sufficient, a large mass of tissue with a high immunological potential was being added simultaneously.

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MATERIALS AND METHODS

Twenty adult mongrel dogs, unrelated to each other, were used in this experiment. This figure does not include those used as splenic and bone marrow donors. These 20 dogs were divided into four groups:

Group A, four dogs, used as controls, were treated with 1000 r T.B.R.[4, 10]

Group B, five dogs were exposed to 1000 r T.B.R. and had a bone marrow homotransplant.

Group C, five dogs were given 1000 r T.B.R. and had a splenic organ transplant in addition.

Group D, six dogs were given 1000 r T.B.R. and had a bone marrow homotransplant and a splenic pulp homotransplant.[1]

Total body irradiation was administered by a C^{137} teletherapy unit delivering 17 r/minute dose rate. The dogs were anesthetized with pentobarbital (Nembutal) and irradiated first on one side and then on the other with a dosage calculated to deliver 1000 r at the midline. The following day, operation or intravenous therapy was performed according to plan.

In the cases of splenic organ transplant, splenectomy was performed on donor dogs under general anesthesia. In the course of this procedure the splenic pedicle was left long. This spleen, thus acquired, was anastomosed to the splenic pedicle of the recipient dog, who had previously been splenectomized, using the Carrell technique with 60 silk in the anastomosis.[8, 13, 17, 18] The success of this technique had been verified by autotransplantation in dogs before this study was begun. Heparin was used early in our experiment to help prevent thrombus formation but was stopped because it seemed that the dangers from postoperative bleeding exceeded those of clot formation.

Bone marrow was procured from the same animal while the dog was still under general anesthesia for splenectomy. The animal was given intravenous heparin, 3 mg./kg., and muscle-splitting incisions were

made over each humerus and femur. A square of cortex was removed from the bone[10] and the marrow curetted and aspirated into the beaker. The aspirate was filtered through a screen (150 holes/inch) fitted into the barrel of a 50 c.c. syringe. The filtrate was given to the recipient dog immediately. Nucleated cell counts were performed on all samples before injection into the dog and varied from 3 to 12 x 10^9 cells.

Splenic pulp was prepared by a method similar to that described by Hume *et al.*[12] Splenectomy was performed and the spleen was immediately cut into small pieces and mashed through a fine tea-strainer with the aid of a glass pestle. The product was suspended in dog's chilled fresh blood and given to the recipient animal as soon as possible. It was noted that the dogs became ill and vomited while receiving this suspension. When this happened the rate of administration was slowed and the dogs did not die. Volumes of spleen given by this method varied from ¼ to ⅝ of a spleen of a dog the same size as the recipient. Donor dogs were females and the recipients in every case were males. In the event of survival after infusion the source of the leukocytes which appeared in the circulation later could then be determined.[6, 16]

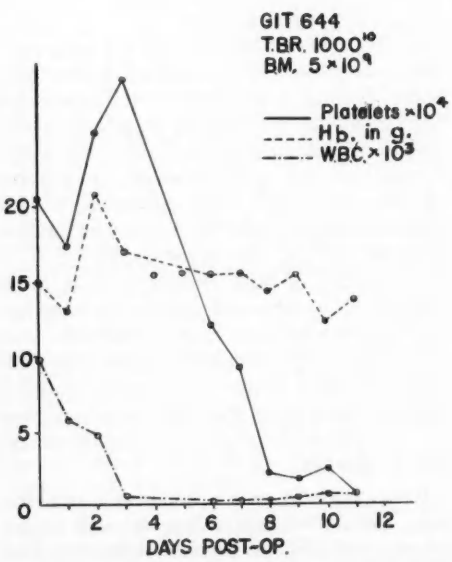


Fig. 1.—Typical blood changes found in a dog who survived for 13 days (Group B).

Results

A. Control Series.—The four dogs receiving only T.B.R. lived three, four, seven and eight days, an average of 5.5 days. The dog that died in three days had developed an intussusception. The others died of the effects of acute radiation injury with early thrombocytopenia and pancytopenia. The bowels of these animals were reddened and edematous. The lungs showed acute congestion with areas of hemorrhage, thrombosis and in some cases bacterial invasion.

B. Total Body Irradiation and Bone Marrow Homotransplant.—The survival time in this group of five dogs was from four to 13 days with an average of 7.4 days. The animal who survived for the longest time had received 1000 r T.B.R. and 5 x 10^9 nucleated bone marrow cells. The hemoglobin and red blood cell count remained reasonably constant during this period. The platelets dropped from a pre-irradiation count of 208,000 to 6500 on the eleventh day. The leukocyte count fell from 9950 to 200 cells on the sixth and seventh day and then rose to 800 to 850 cells terminally. Post-mortem examination showed acute and chronic hemorrhage throughout the entire lung parenchyma and infiltration with acute inflammatory cells. There were many areas of abscess formation and many of the smaller arteries were filled with organized thrombus. Hemorrhage had taken place into the interstitial tissue of the kidneys and extended down into the pelvis. The spleen showed moderate acute and chronic congestion and complete disappearance of lymph follicles. There was mild infiltration of neutrophils throughout the entire specimen and some vacuolation of the red pulp. The liver showed marked central congestion, with marked dilatation of the central veins. There was also some degeneration of the bile ducts. The lymph nodes showed marked acute and chronic congestion. The lymphoid follicles were seen but approximately half the architecture was destroyed.

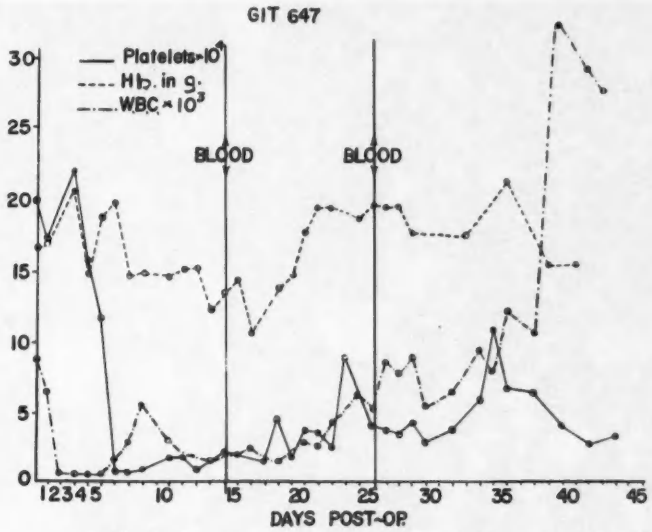


Fig. 2.—Blood changes in the dog, GIT 647, who survived for 43 days (Group D).

The small bowel and adrenal glands were judged to be normal. Death was due to pneumonia (Fig. 1).

C. *Five Dogs with T.B.R. (1000 r), Bone Marrow and Splenic Organ Homotransplants.*—The survival times ranged from three to six days with an average of 4.6 days. In two animals there was an obvious failure of the splenic anastomosis and in all there was necrosis of splenic cells either partial or complete. The leukocyte and platelet counts decreased as in the control group. The lungs showed acute and chronic congestion and the liver showed central congestion.

D. *Six dogs with T.B.R. (1000 r), Bone Marrow and Splenic Pulp Homotransplants.*—The survival times ranged from four to 43 days with an average of 14.2 days. In five of the six dogs there was a typical decrease in the leukocyte and platelet counts with death due to the effects of acute irradiation or to infection. In the dog that survived 43 days a return of circulating neutrophils and platelets occurred. The appetite returned and the dog remained active. Because of a decline in general well-being, the dog was given two transfusions of fresh blood from a male dog, 14 days and 24 days after treatment, but about 30 days after irradiation and effusion the dog began to show evidence of a mouth and middle-ear infection. Cultures taken from these sites grew *staphylococcus aureus* sensitive only to chloromycetin. Despite antibiotics and intravenous therapy the dog died on the 43rd day (Figs. 2 and 3).

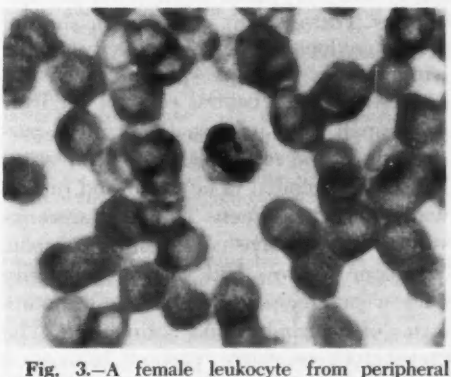


Fig. 3.—A female leukocyte from peripheral blood of the dog, GIT 647, three weeks postoperatively, with its characteristic drumstick — a nuclear projection which has a round-to-ovoid outline and is homogenous in consistency. It is attached by a chromatin strand and is approximately 1.5μ in diameter.

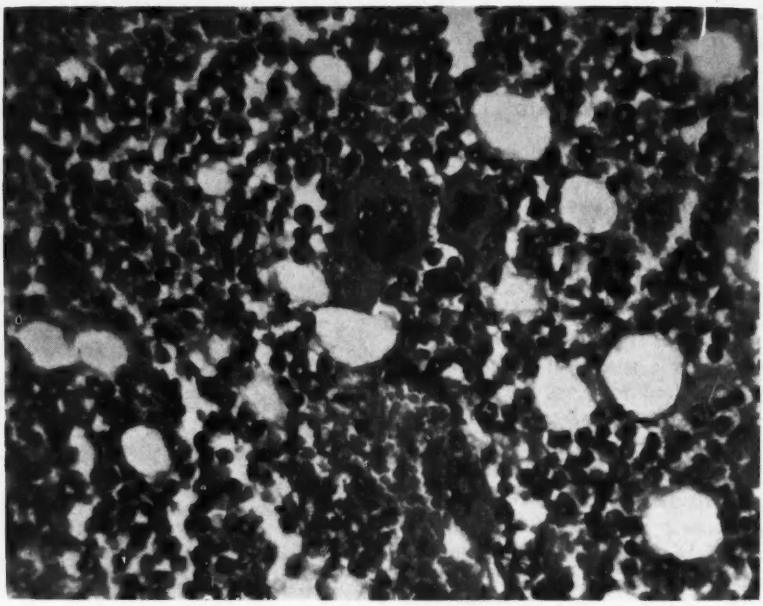


Fig. 4.—Bone marrow of dog, GIT 647, at postmortem showing moderate cellularity.

During the last week of life there was a dramatic elevation of the leukocyte count to a maximum of 38,000 at one time, with a partial return of circulating lymphocytes. Female leukocytes were reported in the peripheral blood in the expected proportion as long as three weeks after irradiation. Further slides taken during the fifth week, however, failed to show female leukocytes. Post-mortem examination showed greatly reduced lymphoid activity in the lymph nodes and the spleen. There was congestion of the central veins of the liver with small areas of hepatic necrosis particularly in the region of the central veins. These areas tended to be linear and run towards the portal tracts. The lungs were involved in a massive confluent pneumonia. The bone marrow had varying cellularity with a predominance of the less mature forms of neutrophils and normoblasts. The megakaryocytes were present in normal numbers (Fig. 4).

Discussion

The only dog that survived for a moderate length of time was a dog that had received both splenic cells and bone marrow. The loss of circulating female leukocytes from the peripheral blood occurred after three weeks and coincided with a dramatic return of the peripheral blood picture to normal and above normal levels. This increase of cells included neutrophils with a pronounced shift to the left as well as lymphocytes up to 10% of the total blood count. The post-mortem bone marrow had varying but definite cellularity while the spleen and lymph glands showed signs of regeneration of lymphocytes.

The most logical conclusion from these observations is that the bone marrow and possibly the splenic cell transplant was a success and that these tissues were destroyed later by the return of the dog's own immune response mechanism.

The other possibility is, however, that the bone marrow and possibly the splenic tissue died out spontaneously after a few multiplications and that this coincided with the return of the dog's hematopoietic function.

Conclusions

The dosage of 1000 r T.B.R. appeared to be lethal to dogs within 10 days. In our hands splenic organ homotransplantation was a hazardous procedure and did not prolong the lives of the dogs exposed to T.B.R. and concomitant bone marrow homotransplantation. The combination of bone marrow and splenic pulp homotransplantation in a dog subjected to 1000 r T.B.R. allowed the animal to live through the acute irradiation phase but did not prevent secondary disease.

This project was supported by National Research Council of Canada Grant No. 713.

Dr. J. S. Thompson, professor of anatomy, University of Alberta, and Dr. M. L. Barr, professor of microscopic anatomy, University of Western Ontario, London, Ont., are thanked for their assistance in the interpretation of sex determination of neutrophils.

Dr. S. R. Usiskin, physicist, Cancer Clinic, The University of Alberta Hospital, Edmonton, is thanked for his assistance in irradiation.

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Résumé

Les difficultés que l'on rencontre lors des tentatives d'homogreffes sont dues principalement à l'existence d'une immunité chez le receveur, provoquant l'élimination du greffon. Des travaux expérimentaux sur ce sujet sont présentés.

Les auteurs utilisèrent une vingtaine de chiens adultes, répartis en quatre groupes:

Groupe A.—Quatre chiens, utilisés comme contrôles, irradiés avec 1000 r.

Groupe B.—Cinq chiens irradiés de la même façon et recevant une greffe de moelle osseuse.

Groupe C.—Cinq chiens, irradiés de la même façon et auxquels on greffa une rate.

Groupe D.—Six chiens, irradiés de la même façon et recevant une homogreffe de moelle osseuse et une transplantation de rate.

Les résultats de ces expériences peuvent être résumés comme suit: dans les séries de contrôle, la survie moyenne fut de 5.5 jours. Les chiens ayant été homogreffés avec de la moelle osseuse eurent une survie moyenne de 7.4 jours. Les chiens du troisième groupe survécurent pendant 4.6 jours. Enfin, dans le dernier groupe, la survie fut de 14.2 jours.

Il semble donc que, d'après ces expériences, les transplantations de moelle osseuse et de rate aient été un succès, et que ces tissus greffés ont été secondairement détruits lors de la réapparition des mécanismes d'immunisation individuels.

CANADIAN JOURNAL OF SURGERY

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A reasonable number of black-and-white illustrations will be reproduced free with the articles. Colour work can be published only at the author's expense. Photographs should be glossy prints, unmounted and untrimmed, preferably not larger than 10" x 8". Prints of radiographs are required and *not the originals*. The magnification of photomicrographs must always be given. Photographs must not be written on or typed on. An identifying legend may be attached to the back. Patients must not be recognizable in illustrations, unless the written consent of the subject for publication has been obtained. Graphs and diagrams should be drawn in India ink on suitable white paper. Lettering should be sufficiently large that after reduction to fit the size of the Journal page it can still be read. Legends to all illustrations should be typed separately from the text and submitted on a separate sheet of paper. Illustrations should not be rolled or folded.

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The Royal College of Physicians and Surgeons of Canada

NEWSLETTER

In the April Newsletter, mention was made of the forthcoming visits of two travelling professors for the Fall of 1961. Itineraries for their respective tours are as follows:

Sir George Pickering, Regius Professor of Medicine, Oxford University, R. S. McLaughlin-W. E. Gallie Visiting Professor for 1961: Montreal, October 4-16; Toronto, October 17-20; London, October 23-November 3; Saskatoon, November 7-9; Regina, November 10, and Montreal, November 12-14.

Sir George will also pay brief visits to Ottawa and Hamilton from Montreal and Toronto respectively.

Dr. Paul Wood, Physician to the National Heart Hospital, London, 1961 Sir Arthur Sims Commonwealth Travelling Professor to Canada: Vancouver, October 1-4; Edmonton, October 4-6; Winnipeg, October 15-20; Toronto, October 22-28; Kingston, October 29-November 1; Ottawa, November 2-4; Halifax, November 5-10, and Montreal, November 12-18.

The influence of such outstanding men will continue long after their short visits to Canada come to an end.

The President of The Royal College, Dr. D. A. Thompson, and Council Member, Dr. D. R. Webster, represented the Fellows of the Surgical Division of the College at the meeting of the International Federation of Surgical Colleges at Oslo, Norway, in September 1961. The aim of this new Federation is to improve the standard of surgical teaching and training throughout the world.

Much interest has resulted from the announcement of the Federal Government concerning the Royal Commission on Health Services. In January 1961, the Council of The Royal College authorized the Executive Committee to initiate whatever steps it considered necessary to prepare a Brief to the Royal Commission, confining itself, as an educational body, to matters relating to the supply and distribution of medical specialists to meet the present and future needs of the Canadian people. As well as this, the Brief was to stress methods to assure facilities for the training of such specialists to meet these needs. In any scheme where continuing improvement in the standard of graduate medical education and the quality of medical care is to be attained, the importance of research and the development of scientific medicine in general must be recognized. To obtain some basic facts, the Council, at its June meeting, appointed Dr. John W. Scott of Edmonton to gather all available information on the present and future supply and distribution of specialists.

At its June meeting, Council approved the establishment, commencing in 1962, of a Fellowship Examining Board in Obstetrics and Gynecology which will report directly to the Committee on Examinations in the same manner as the Fellowship Examining Boards in Medicine and in Surgery. It was felt that this action is consistent with the individual status enjoyed by this branch of medicine in most countries throughout the world. The action was taken in accord with the expressed wishes of the third largest group of Fellows in the College.

New regulations relating to training requirements and examinations for Fellowship in Surgery as modified for Cardiovascular and Thoracic Surgery were approved at the June meeting of Council. These are:

REGULATIONS RELATING TO THE TRAINING REQUIREMENTS: FELLOWSHIP EXAMINATION IN SURGERY (MODIFIED FOR CARDIOVASCULAR AND THORACIC SURGERY)

The cardiovascular and thoracic surgeon has special need of a complete knowledge of the physiology of the cardiovascular and respiratory systems. He should possess a broad knowledge of dis-

eases of the chest and cardiovascular system.

In some centres experience in the surgery of the lungs and other structures within the thorax may be obtained on the same service with the cardiovascular surgery; in others, this experience will be gained as part of the general surgical training.

During the course of training the candidate must acquire a knowledge of the basic sciences necessary to the understanding and practice of cardiovascular and thoracic surgery. In addition to the specific requirements detailed in section 2(c) of the regulations hereafter, further training in the basic sciences (such as anatomy, physiology, biochemistry and pathology) may be taken concurrently with the resident training, by attendance at special courses in the basic sciences.

The requirements as set forth below are considered to be minimal training for the field of cardiovascular and thoracic surgery. It is recommended that candidates take additional training, which could be in autopsy pathology, cardiology, internal medicine, related basic science or further clinical study in the specialty in Canada or abroad.

1. An approved general interneship of at least one year.
2. Four years of graduate training in addition to the general interneship. This *must* include:
   (a) One year of approved resident training in general surgery (read preamble).
   (b) Two years of approved resident training in cardiovascular and thoracic surgery. The candidate's training must include the equivalent of six months' approved resident training in surgery of the thoracic wall, mediastinum, esophagus and diaphragm, as well as the lung, and not less than one year of approved resident training in cardiovascular surgery. During the course of this training, adequate experience must be obtained in the management of both congenital and acquired lesions.
   (c) One year of approved laboratory training in the techniques of cardiovascular surgery, extracorporeal circulation and hemodynamics. At least six months of this year must be spent exclusively in the laboratory; the remainder of the year may be spent in the laboratory concurrently with resident training in the specialty where the training program is organized to provide this dual experience.

REGULATIONS RELATING TO THE EXAMINATIONS:
FELLOWSHIP EXAMINATION IN SURGERY
(MODIFIED FOR CARDIOVASCULAR AND THORACIC SURGERY)

The examination will consist of a written examination and an oral and clinical examination.

(a) The *written examination* will consist of three papers:
   Paper No. 1. The principles and practice of surgery with emphasis on cardiovascular and thoracic surgery.
   Paper No. 2. Clinically applied pathology and bacteriology with emphasis on the application of these sciences to cardiovascular and thoracic surgery.
   Paper No. 3. Clinically applied basic sciences including anatomy, physiology and biochemistry or other appropriate basic sciences with emphasis on the application of these sciences to cardiovascular and thoracic surgery.
   In each of the examination papers one question will be derived from the general surgical field while the remaining three questions will deal directly with the field of cardiovascular and thoracic surgery.

(b) The *oral and clinical examination* will consist of two parts:
   (1) A clinical examination conducted by a Board of Examiners consisting of at least two surgeons trained in the field of cardiovascular and thoracic surgery and one general surgeon. The candidate will be asked questions dealing with the broad fields of surgery and cardiovascular and thoracic surgery, and the related basic sciences; and in addition will be examined on assigned patients suffering from cardiovascular or thoracic surgical conditions.
   (2) An oral examination in applied pathology and bacteriology conducted by a Board of Examiners representing surgery, pathology and cardiovascular and thoracic surgery. Emphasis will be placed on the principles of pathology and bacteriology, pathogenesis, pathological physiological changes and their clinical application to cardiovascular and thoracic surgery. The

candidate will be expected to demonstrate a broad knowledge of gross pathology: relatively less stress will be placed on the recognition of histological sections and technical bacteriology, but recognition of microscopic sections of common surgical lesions and of cardiovascular and thoracic lesions will be required.

It should be noted that the "modified" examinations for certification in the following surgical specialties *have* been discontinued: Neurosurgery, Obstetrics and Gynecology, Otolaryngology and Plastic Surgery. This means that all candidates for certification in these specialties despite holding other recognized qualifications must take the full examination both written and oral.

The Council takes pleasure in announcing the election to Fellowship under Article 3, Section 3, of the Regulations, of Dr. Frank Turnbull of Vancouver.

Attention is drawn to an announcement in the next column of the Annual Meeting of The Royal College of Physicians and Surgeons of Canada to be held in Toronto in January 1962. This year, certified specialists are invited to attend the scientific sessions subject to the advance registration as noted in the application form.

The Council wishes to acknowledge the generous gift of the Medical Council of Canada of $15,000 to be used for furnishing the Archives Room in the College Building as a memorial to the founder of the Medical Council of Canada, Sir Thomas Roddick, M.D., C.M., LL.D. (Edin.), F.R.C.S.(Eng.). The Archives and Library Committee, under the Chairmanship of Dr. D. Sclater Lewis, has been asked to undertake the planning and furnishing of the room in consultation with the Chairman of the Building Committee, Dr. George Hooper. A portrait of Sir Thomas hangs in the Archives Room on permanent loan from the Montreal General Hospital.

W. Gordon Beattie, F.R.C.S.[C],
Honorary Assistant Secretary
September 8, 1961.

SASKATCHEWAN REGIONAL MEETING

The Saskatchewan Regional Meeting of The Royal College of Physicians and Surgeons of Canada will be held in Regina, Saskatchewan, on Thursday and Friday, November 23 and 24, 1961. This is the third of such regional meetings sponsored by The Royal College in its program to provide additional educational opportunities to members of the medical profession engaged in specialist practice.

A cordial invitation is extended to Fellows and Certificated Specialists of The Royal Collegs of Physicians and Surgeons of Canada living in Saskatchewan to attend this meeting. Specialists in adjacent areas of Manitoba and Alberta are also invited to attend.

The meeting will be divided into three sections — Medicine and Pediatrics, Obstetrics and Gynecology, and Surgery, and will be held at the Hotel Saskatchewan. The program will include presentations by a number of highly qualified guest speakers.

INVITATION TO CERTIFICATED SPECIALISTS OF THE ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA TO ATTEND THE 1962 ANNUAL MEETING OF THE COLLEGE

In 1959, the College, in keeping with a policy of expanded educational opportunities for Fellows and certificated specialists, embarked on a program of Regional Scientific Meetings, which Fellows and certificated specialists living in the designated region have been invited to attend.

At the 1961 Annual Meeting of the College held in Ottawa, certificated specialists in the immediate local area were invited to attend the scientific program sessions. Because of the larger meeting-room accommodation available in Toronto, the Council of the College has decided to extend an invitation to all certificated specialists of The Royal College to attend the scientific sessions at the 1962 Annual Meeting, to be held at the Royal York Hotel, Toronto, from January 18 to 20.

Certificated specialists wishing to attend this meeting must complete the registration application form overleaf and return it to The Secretary, The Royal College of Physicians and Surgeons of Canada, 74 Stanley Ave., Ottawa 2, Ont., together with a cheque or money order in payment of the registration fee of $15.00, made payable to the Royal College of Physicians and Surgeons of Canada.

The Secretary,
The Royal College of Physicians and Surgeons of Canada,
74 Stanley Avenue,
Ottawa 2, Ontario.

I desire to register to attend the Scientific Sessions of the Annual Meeting of The Royal College of Physicians and Surgeons of Canada to be held at The Royal York Hotel, Toronto, January 18, 19 and 20, 1962.

Enclosed is a cheque/money order in the amount of $15.00 in payment of the Registration Fee.

Name of Certificant: .....

Address: .....

.....

Name of Specialty: .....

(please print)

Letter to the Editor

THE FIRST APPENDECTOMY

To the Editor:

In the article on Abraham Groves and the first elective appendectomy (*Canad. J. Surg.*, 4: 405, 1961), reference was made to Mr. John A. Shepherd's article on "Acute Appendicitis, a Historical Review" (*Lancet*, 2: 299, 1954). There, Mr. Shepherd stated that Lawson Tait was probably the first to diagnose and remove an acutely inflamed appendix in Great Britain, but he was unable to find the record. Subsequently he found it in the *Birmingham Medical Review* (27: 27, 1890) and he reported this in the *Lancet*, 2: 1301, 1956. This had escaped my notice. Lawson Tait first diagnosed and successfully removed an acutely inflamed appendix in 1880 and operated on a second case in 1886. His first case preceded that of Groves by three years. Subsequently, he stated that the risk of operation was increased by the detail of removal of the appendix and he reverted to a conservative drainage procedure. Tait thought that right-sided inflammation in the abdomen was often due to typhoid fever, but he recognized increasingly the high proportion of cases of typhlitis that were due to primary disease of the appendix. I am again indebted to Mr. Shepherd.

Dr. E. H. Bensley of Montreal has kindly provided an additional list of Groves' writings.

In the *Canadian Journal of Medical Science*:

A simple aspirator, 2: 109, 1877

Supra-pubic lithotomy, 6: 354, 1881

In the *Canadian Practitioner*:

Removal of a firmly adherent, solid tumour of the ovary, 8: 323, 1883

The operative treatment of fluid effusions in the chest, 9: 233, 1884

Prostatotomy, 12: 240, 1887

Peri-typhlitic abscess, 15: 394, 1890

The local treatment of intra-uterine sepsis, 24: 134, 1899

Screw nails in fractures, 26: 25, 1901

Roentgen rays in relation to cancer, 27: 313, 1902

The diagnosis of stone in the bladder, 27: 493, 1902

C. W. Harris, M.B., F.R.C.S.

Room 342,
Professional Suites,
Toronto Western Hospital,
Toronto 2B.

FORTHCOMING MEETINGS

AMERICAN COLLEGE OF SURGEONS –FIRST 1962 SECTIONAL MEETING

Surgeons, graduate nurses, and all related medical personnel are invited to the first of four 1962 Sectional Meetings of the American College of Surgeons in Los Angeles, January 29 to February 1. Headquarters hotel for the doctors' sessions will be the Statler-Hilton, and for nurses, The Biltmore.

This is the only four-day meeting for 1962 scheduled by the College, and the only meeting with a special program for nurses.

Scope of this meeting approaches that of the annual Clinical Congress. Doctors' sessions will be held in general surgery and the specialties of obstetrics and gynecology, ophthalmology, otolaryngology, urology, neurological surgery, plastic surgery, pediatric surgery and thoracic surgery. There will be daily "How I Do It" non-operative clinics — educational demonstrations by surgeons noted for specific techniques; 38 scientific papers; 28 panel discussions; 11 symposia, as well as selected films and industrial exhibits of interest.

Dr. William P. Longmire, Jr., professor and chairman, department of surgery, University of California, is chairman of the local advisory committee on arrangements.

Other Sectional Meetings during 1962 will be held in Detroit, March 5-7; Memphis, March 26-28, and Washington, D.C., April 16-18.

FIRST INTERNATIONAL CONFERENCE ON ORAL SURGERY

The First International Conference on Oral Surgery, sponsored by the American Society of Oral Surgeons in conjunction with the Royal College of Surgeons of England, will be held at the Royal College in London, July 1-4, 1962. All interested dentists and physicians are invited to attend, and a bulletin describing the Conference is available on request from the American Society of Oral Surgeons, 840 North Lake Shore Drive, Chicago 11, Ill. The program will include symposia on the temporomandibular joint and on maxillofacial injuries, in addition to a wide variety of scientific papers and discussions. Approximately one-half of these papers will be presented by essayists from North, South and Central America and the other half from the United Kingdom, Europe, Africa and Asia. Visitors attending the Conference will also have an opportunity to observe surgical procedures in the hospitals of London as a part of the Conference program.

Extensive social and ladies' programs have been arranged by the Joint British-United States Committee. Features of the social program will be a banquet at London's famed and historic Guildhall and a reception by H.M. Government.

For additional details and bulletins, dentists and physicians in the United Kingdom and Europe should write to Dr. Terence Ward, c/o Royal College of Surgeons, Lincoln's Inn Fields, London, W.C.2. Dentists and physicians in the U.S. and other countries should write to Mr. D. C. Trexiler, American Society of Oral Surgeons, 840 N. Lake Shore Drive, Chicago 11, Illinois, U.S.A.

NOTICE

FORMATION OF THE CANADIAN ASSOCIATION OF GASTROENTEROLOGY (L'ASSOCIATION CANADIENNE DE GASTROENTEROLOGIE)

On June 20, 1961, the organizational meeting of the Canadian Association of Gastroenterology (l'Association Canadienne de Gastroentérologie) was held at the Queen Elizabeth Hotel in Montreal. This bilingual society was brought into being as the result of a nationwide survey among many of those physicians and surgeons actively engaged in this subspecialty in Canada who felt that such a group could make a needed and valuable contribution toward encouraging and engaging in research, study and practice in this field. This society would also serve as a means of communication between gastroenterologists and scientists in allied fields related to gastroenterology in Canada.

Officers elected for the year 1961-62 are as follows: Dr. R. D. McKenna, Montreal, President; Dr. R. C. Dickson, Halifax, President-Elect; Dr. Walter MacKenzie, Edmonton, Vice-President; Dr. Ivan T. Beck, Montreal, Secretary; Dr. Douglas G. Kinnear, Montreal, Treasurer. Councillors: Dr. A. Bogoch, Vancouver; Dr. Malcolm Brown, Kingston; Dr. Paul Letendre, Montreal; Dr. R. M. MacDonald, Halifax; Dr. Eric Nanson, Saskatoon, and Dr. K. J. R. Wightman, Toronto.

At present in the process of incorporation, this new society will hold its next annual meeting in Winnipeg in June 1962, at which time scientific papers will be presented by members and invited guests at its one-day session.

BOOK REVIEWS

(See also page 592)

SURGERY IS DESTINED TO THE PRACTICE OF MEDICINE. 90th Hunterian Oration, Royal College of Surgeons of England, February 16, 1959. Sir Reginald Watson-Jones. 81 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $3.60.

John Hunter died in 1793 leaving an estate which consisted chiefly of his incomparable collection of anatomical and biological specimens on which he had expended more than £70,000. Six years after his death, the Government of Great Britain reluctantly purchased it for £15,000. The responsibility of caring for it was refused successively by the Royal College of Physicians, the Royal Society and the British Museum. In 1799 the Corporation of Surgeons agreed to accept it and to be responsible for its maintenance. One year later, in 1800, the Corporation of Surgeons became the Royal College of Surgeons of England. During the 161 years which have elapsed since then, Hunter and his ideals have been the stimulus for much of the productive activity of this great centre of surgical education and the Hunterian Museum has become one of the greatest assets of the College.

In 1813, the two trustees of Hunter's estate, his nephew Matthew Bailey and his brother-in-law Sir Everard Home, inaugurated within the Royal College of Surgeons of England the institution of The Hunterian Oration to be delivered annually on February 14, which was Hunter's birthday. It was delivered annually from 1814 to 1853 and since then, every other year, by the most distinguished surgeons of Great Britain. Over the years the Hunterian Oration has kept green the memory of John Hunter and his great work, and it has become an accolade of distinction for those chosen to be the Hunterian Orator.

Sir Reginald Watson-Jones delivered the 90th Hunterian Oration in 1959 and his Oration is published in this volume. The somewhat cryptic title was chosen to emphasize the fundamental principle that surgery is a discipline dedicated to the cure of disease; that a surgeon must be a physician in his approach to the study of human disease; a special type of physician who uses surgical methods for the alleviation or cure of disease; that the practice of surgery is a craft no longer but has become an art fundamentally concerned with the nature of living organisms and deviations from their normal structure and function. Treatment of disease by surgical methods is secondary to the search for knowledge of the disease as a whole. This was Hunter's approach to surgery and his principle that surgery was the last resort in the treatment of disease has become of more importance as our knowledge of disease is increased.

Sir Reginald Watson-Jones has drawn on his wide knowledge of orthopedic problems to illustrate these principles and to indicate the progress which has been made in our generation towards improved knowledge of disease and the elimination of many causes of crippling, formerly commonplace. He includes many examples from Dame Agnes Hunt's case books, hitherto unpublished.

This is a stimulating and interesting monograph which continues the high tradition of Hunterian Orations. It reminds us that today, as always, the search for knowledge is the foundation of progress in medicine. That was the principle to which John Hunter was dedicated and it is the foundation of his greatness. It is a principle even more fundamental today than when John Hunter lived.

SURGERY OF THE ESOPHAGUS. Raymond W. Postlethwait and Will Camp Sealy. 482 pp. Illust. Charles C Thomas, Springfield, Ill.; The Ryerson Press, Toronto, 1961. $33.00.

This is a most excellent book by two surgeons from Duke University Medical Center, Durham, North Carolina. It is written for surgeons, but it is so comprehensive and so impartial that it will be of almost equal value to others, such as gastroenterologists, otolaryngologists and pathologists. The opinions expressed in the book are based on a reasonably complete survey of the relevant literature, supplemented by a careful study of all cases of esophageal disease treated at Duke University since 1930, and brought into sharp focus by relating them to the authors' own extensive personal experience. Each major topic is dealt with thoroughly under all the usual headings. Special commendation is earned by the inclusion in each chapter of a section on historical aspects, and also by the careful data on operative results, mortality and survival statistics (both the authors' own and the cumulated figures from other published works). All accounts are fully illustrated with photographs of roentgenograms, pathological specimens and microscopic sections, mostly from the authors' own cases. Major operative procedures are delineated by large clear drawings. References, including titles (to a total of about 3500), are listed alphabetically at the end of each chapter. Particularly good is the chapter on chemical burns of the esophagus, which includes a description of the newer techniques for esophageal reconstruction and bypass. Also praiseworthy are the chapters on squamous carcinoma of the esophagus and on esophageal varices.

The whole book is nicely written and beautifully printed. It is highly recommended to all who have an interest in the esophagus and its diseases.

ROENTGENOLOGY OF INTRACRANIAL MENINGIOMAS. Sidney P. Traub, Associate Professor of Radiology, University of Saskatchewan College of Medicine. 238 pp. Illust. Charles C Thomas, Springfield, Ill., 1961. $14.00.

This monograph is a review of 170 cases of meningiomas seen at the Montreal Neurological Institute in the years from 1934 to 1953. The book is pleasing in appearance and the material neatly set out for ease of reading on good quality paper. The excellence of the illustrations is as much a tribute to the radiographers as to the publisher, from whom we have come to expect work of this quality.

The greater, and by far the best part of the book is devoted first, to the plain film changes found in intracranial tumours, and then specifically in the meningioma according to its site. The main findings are hyperostosis, abnormal skull vascularity, bone destruction and tumour calcification; the last is uncommon in the posterior fossa meningioma. The author stresses that abnormalities which suggest the presence of a tumour were found in 88% of the cases; in 33.5% these were diagnostic of meningioma, while in only 21 were the films considered to be normal. This indicates how rewarding a careful study of the plain films may be.

The book goes on to discuss the encephalographic findings of the supratentorial meningiomas in some detail. However, the full value of encephalography has not been exploited. The olfactory cisterns are not mentioned in the text, but Fig. 95a is a beautiful example of displacement of these cisterns by a meningioma of the tuberculum sellae. The posterior fossa meningiomas are only briefly considered, yet they form a very important group where precise anatomical localization is required by the surgeon and can be achieved by encephalography.

It is unfortunate that only seven of the tumours were investigated by angiography. Instead there is an excellent review of Wickbom's 84 cases, in which illustrations of 17 cases seen by the author at the University Hospital, Saskatoon, are used.

The book concludes with a descriptive summary and bibliography. It should prove of interest to the radiologist, neurologist and neurosurgeon.

THE SURGERY OF MITRAL STENOSIS. Robert P. Glover and Julio C. Davila, University of Pennsylvania School of Medicine, Philadelphia. 219 pp. Illust. Grune & Stratton, Inc., New York; The Ryerson Press, Toronto, 1961. $10.50.

This monograph is a thorough analysis of over 1000 cases that have been operated upon by Dr. Glover.

The strongest and most informative chapter is that dealing with the anatomy and pathology of the mitral valve. A most lucid description of a complex anatomical structure is given.

The description of the technique of mitral commissurotomy may be disappointing to those who advocate transventricular valvotomy. The technique of commissurotomy, which the authors have clearly mastered, cannot be wholeheartedly recommended for use by less experienced surgeons as it carries a greater risk of postoperative mitral insufficiency than do the conventional techniques of finger fracture or transventricular valvotomy.

The surgical technique of the operation treatment of recurrent stenosis and the paragraphs dealing with open operation for mitral stenosis are not extensive.

However, the complications of commissurotomy, including restenosis and the production or aggravation of mitral insufficiency, are adequately discussed.

The book is complemented by an appendix in which graphic comparison of extensive data is presented. This, in the reviewer's opinion, is one of the most worthwhile parts of the book.

In summary, this is a comprehensive and well-written monograph of interest to surgeon and physician alike, and is recommended to those interested in this facet of medicine.

CLINICAL ORTHOPAEDICS. Editor-in-Chief Anthony DePalma, with the assistance of the Associate Editors, the Board of Advisory Editors and the Board of Corresponding Editors. No. 18. 293 pp. Illust. J. B. Lippincott Company, Philadelphia and Montreal, 1960. $6.00 to sustaining members, $7.50 per individual copy.

This is a triannual publication produced under the auspices of the Association of Bone and Joint Surgeons. The book is printed in a double-column style for easy reading, and has a hard cover. The size and quality of production make it a handsome addition to the library shelf. The book has three sections. The first section is designed to cover a specific subject, in this instance, Internal Derangements of the Knee Joint, in a complete manner. Many of the articles in this section have been previously presented by the author; for example, Arthrodesis of the Knee by Charnley; while others, such as Tibial Plateau Prosthesis by McKeever, represent new material. In general this section is a textbook-type coverage of a subject by outstanding authors and has much to commend it to surgeons working in this field. The second section is devoted to subjects of general orthopedic interest in standard journal fashion, and the quality of the articles varies considerably. The third section entitled "Items" contains articles on miscellaneous orthopedic topics, and one questions the necessity of its segregation from section two.

The reviewer was impressed by the quality of this publication and recommends it to those interested in orthopedic surgery.

ABDOMINAL SURGERY. Edited by Arthur W. Allen and David Woolfolk Barrow. 658 pp. Illust. Paul B. Hoeber, Inc., New York, 1961. $21.50.

One-volume, multiple-author textbooks of surgery confined to one specialty may be the coming thing because surgical knowledge has become so broad and so specialized that the whole field can no longer be covered properly by one individual. This book contains over 600 pages and 542 illustrations and is the work of 41 authors. It is dedicated to Dr. Allen, who died before its publication, and represents the teaching and practice in the United States of many of its best-known and respected general surgeons.

Only such a great teacher could gather together such authorities as the writers of this book: a foreword by Frederick Coller; preoperative preparation by Maddock and Clarke; anesthesia by Adriani; postoperative care by Ochsner and Blalock; gastric surgery by Marshall and Adams; gallbladder by Glenn; bile ducts by Waltman Walters; pancreas by Elliott, Williams and Zollinger; diverticulitis by Judd and Waugh; ulcerative colitis by Cave and Thompson, and rectum and anus by Turnbull and Crile. These are examples of the impressive list of contributors.

The student of surgery, of whatever vintage, will be interested in this presentation of the present accepted practice of abdominal surgery in America. The candidate for postgraduate examinations will not be led astray, and the experienced surgeon will be interested in reading the present teaching on the lesions and techniques as practised by the great surgeons south of the border. It is highly recommended to all.

LE KYSTE HYDATIQUE (THE HYDATID CYST). Thérapeutique chirurgicale. P. Goinard, Professeur de Clinique thérapeutique chirurgicale, Faculté de Médecine d'Alger, J. Pegullo, Chirurgien des Hôpitaux d'Alger, et G. Pélissier, Chirurgien-assistant des Hôpitaux d'Alger. 204 pp. Illust. Masson & Cie, Paris, 1960. 45 NF. $9.00 (approx.).

Les auteurs ont le mérite de présenter en une monographie toutes les localisations de l'hydatidose humaine: foie, poumons, péritoine, plèvre, médiastin, paroi thoracique, diaphragme, cœur, cerveau, tête et cou, rachis, rate, reins, pancréas, os et muscles.

Il est curieux de constater que, malgré l'absence presque complète de cette affection dans nos régions, nos connaissances sont assez à point, du moins sur l'hydatidose des grands organes. Il est vrai que c'est le professeur qui parle.

Les auteurs ne manquent pas de signaler l'étonnante vie alternée des parasites dans deux hôtes successifs d'espèce différente (l'un carnivore, l'autre herbivore), et ensuite l'homme qui "entre en dérivation sur ce cycle" parce qu'omnivore.

Il serait hors d'une analyse de vouloir résumer le comportement de l'hydatide au niveau de chaque organe, comme le font si bien les auteurs, mais contentons-nous d'en signaler les deux formes extrêmes: la volumineuse boule d'eau solitaire dans les viscères, les innombrables et minuscules grains vésiculaires des os et enfin toutes les formes intermédiaires.

Les auteurs, dans un volume de 200 pages avec de merveilleuses illustrations, nous font profiter de leur féconde expérience dans le milieu algérien où l'hydatidose est monnaie courante.

Il est bien difficile pour nous, avec nos seules données théoriques, de faire une critique adéquate de cet ouvrage tant au point de vue médical que chirurgical.

De toute façon, les auteurs ont certainement comblé une lacune dans la littérature médicale.

GESCHICHTE DER ORTHOPAEDIE (HISTORY OF ORTHOPEDICS). Bruno Valentin. 304 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $9.90.

It is surprising to note in the introductory remarks of the author that this is the first book ever published on the history of orthopedics in any language.

Pathological changes have been frequently observed in ancient bone fragments, skeletons, mummies and even in the remains of Neanderthal man. Prehistoric man suffered from the same destructive, degenerative and infectious diseases that cause deformities in our time. Tuberculosis, gibbus formation, dysostosis and chondrodystrophies are clearly identifiable in these prehistoric skeletal remnants. Such deformities, being easily observable and likely to interfere seriously with the harsh and active life that was the lot of prehistoric man, no doubt became the early objects of the healing art.

This book is divided into two parts. The first portion contains chapters on the history of the most common orthopedic disorders and the development of their treatment. The second part outlines the historical development of the science and art of orthopedics in the various countries that comprise so-called Western civilization. From the historical viewpoint it would have been interesting to read about the influence and contributions of the Far East and especially of Arabian medicine.

Nothing is more difficult than a beginning and this applies to the effort of the author. He had to produce a book which required years of study and collecting of data. This reviewer thinks that he has succeeded admirably.

(Continued on page 608)

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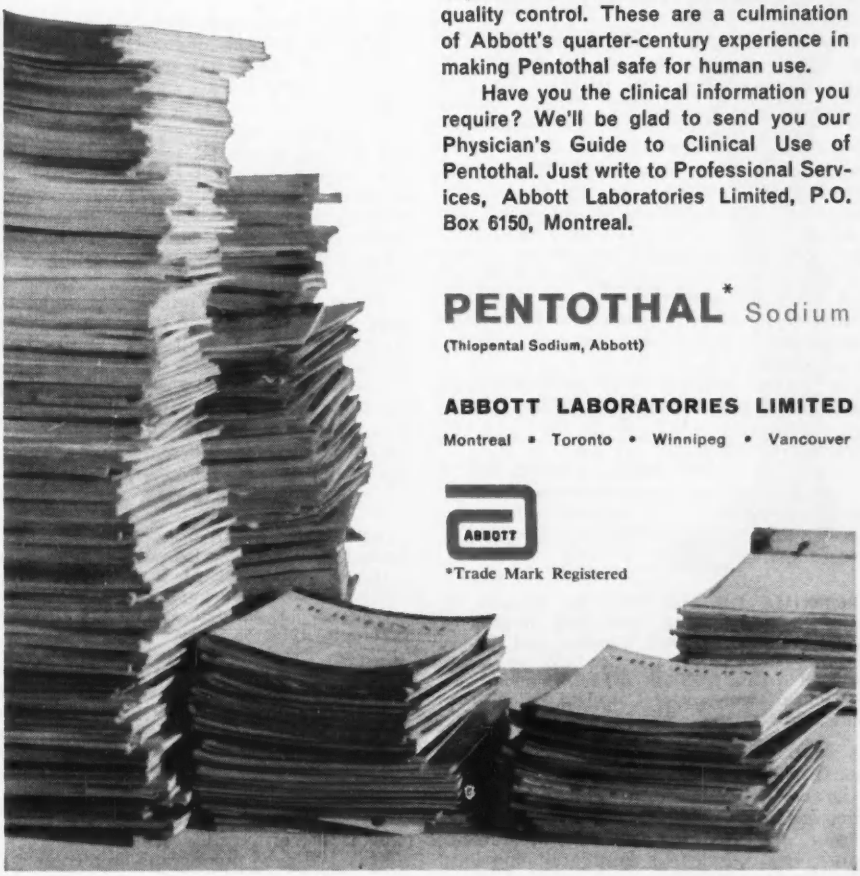
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(Continued from page 606)

INTRA-ABDOMINAL CRISES. Kenneth D. Keele and Norman M. Matheson, Ashford Hospital, Middlesex. 397 pp. Illust. Butterworth & Co. (Canada) Ltd., Toronto, 1961. $10.00.

Abdominal emergencies are commonly regarded as lying chiefly within the province of the surgeon, but there are many acute conditions which arise within the abdomen which do not necessarily require operative intervention. The second class of abdominal emergencies give rise to difficulties in the differential diagnosis. This book represents a successful attempt on the part of two experienced clinicians, one an internist and the other a surgeon, to survey the field of acute abdominal conditions which occur in practice. In the authors' own words, they set out to "resolve the different attitudes of physician and surgeon into urgent medico-surgical synthesis". Such an attitude, which already exists within other clinical fields, is to be welcomed and commended.

The book is divided into three parts. The authors first discuss the general features of intra-abdominal crises and consider the presenting symptoms and signs. In the second part, local abdominal diseases are considered, the majority of which are surgical in nature; they include acute vascular, urological and gynecological conditions, in addition to the many local gastrointestinal lesions which are of an emergency nature. The third part deals with acute intra-abdominal conditions arising as a part of a more general disease; these fall more naturally within the province of the physician. However, they often cause the surgeon concern when he is faced with a problem in diagnosis.

The authors conclude with an appendix in which they discuss the value of various laboratory investigations which can be used in acute abdominal conditions.

The book is to be heartily recommended to all young physicians and surgeons. Furthermore, it should be a welcome addition to the bookshelves of their more senior colleagues in both disciplines.

CARCINOMA OF THE COLON. Edward G. Muir, Surgeon to H.M. Household, Surgeon to King's College Hospital, London, and Queen Victoria Hospital, East Grinstead. 181 pp. Illust. Edward Arnold (Publishers) Ltd., London; The Macmillan Company of Canada Limited, Toronto, 1961. $7.15.

This short simple book is aptly directed to the surgical house officer. In discussing carcinoma of the colon, exclusive of the rectum, the author has set himself a difficult and, to this reviewer, unnatural task. Even so, the subject is large, the coverage is brief and omissions are inevitable. He obviously speaks from a wealth of clinical experience and his management of practical problems is the best feature of the book. The chapters include those on surgical anatomy, pathology and diagnosis, but the chief emphasis (half of the book) is on the operations for carcinoma of the colon. The book could be improved by editing to eliminate spelling errors and paragraph-like sentences. The illustrations on the whole are good, with many line drawings of operative procedures. Occasionally the drawings are complex, as on page 117 when quite different procedures for a tumour of the upper rectum and lower sigmoid, only centimetres apart, are outlined. There is a good bibliography.

CONGENITAL DEFORMITIES. Gavin C. Gordon, Consultant Orthopedic Surgeon, Cumberland and North Westmorland; Orthopedic Surgeon and Adviser in Physical Medicine, Irton Hall School (N.S.S.), Cumberland, England. 128 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $6.35.

This book attempts to apply experience, scholarship, and the scientific method to the understanding of congenital anomalies. The author deserves credit for his success in this subject which has not yet been explored intensively.

A discussion of dislocation of the hip forms the core of the book, and its relationship to other hip conditions, e.g. Legg-Calvé-Perthe's disease, is considered. Several important details of congenital luxation, such as anteversion, are convincingly delineated. The book is a valuable index because of its complete references.

There are two appendices; "A" which deals with osteitis and with the McMurray osteotomy, and "B" which discusses cerebral palsy. "A" is particularly interesting and thought-provoking, "B" less so. The many illustrations and radiographic pictures are well reproduced. The style of the book is a difficult one because of lack of simplicity. I recommend this text to all orthopedic surgeons.

CARDIAC SURGERY 1960-61. Edited by Charles P. Bailey, Chairman and Professor of Surgery, New York Medical College. 179 pp. Illust. Davis Monograph Series. F. A. Davis Company, Philadelphia; The Ryerson Press, Toronto, 1960. $5.50.

This monograph written by Dr. Bailey and his associates deals with nine topics commonly encountered in cardiac surgery. The material on coarctation, ventricular septal defects, aortic stenosis and aortic insufficiency is clearly presented and represents the standard concepts on these subjects.

The monograph is well illustrated and the index is well arranged.

This 179-page monograph is recommended as a useful reference source for medical students, cardiac surgeons and cardiologists.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Recurrent Dislocation of the Shoulder. H. F. Moseley, Director, Accident Service, and Surgeon, Royal Victoria Hospital; Associate Professor of Surgery, McGill University, Montreal. 163 pp. Illust. (including plates and stereoscopic photographs). McGill University Press, Montreal, 1961. $13.50.

The Shaking Palsy (Parkinson's Disease): A Symposium. Conference Editors, Harold Elliott and Blaine Nashold. 160 pp. Illust. McGill University Press, Montreal, 1961. $5.00.

Some Aspects of Obliterative Vascular Disease of the Lower Limb. J. A. Gillespie and D. M. Douglas. 136 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $5.00.

A Synopsis of Ophthalmology. 2nd ed. J. L. C. Martin-Doyle. Foreword by H. B. Stallard. 249 pp. Illust. John Wright and Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1961. $4.70.

Thrombosis and Anticoagulant Therapy. Proceedings of a Symposium arranged by Professor P. A. Owren, Professor R. B. Hunter and Dr. W. Walker and held in Queen's College, Dundee, on 29 and 30 September and 1 October, 1960. Edited by W. Walker. 106 pp. Illust. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Limited, Toronto, 1961. $3.00.

Traité de technique chirurgicale. 2nd ed. in 8 Vols. Edited by Marcel David. Vol. III. Crâne, Encéphale, Rachis, Moelle. Part I. Généralités, méthodes d'exploration. 698 pp. Illust. Masson & Cie, Paris, 1961. 132 NF. $26.40 (approx.).

Varicose Veins: A Practical Manual. R. Rowden Foote, London; 3rd ed. with the assistance of A. Gordon Dingley. 356 pp. Illust. John Wright & Sons Ltd., Bristol; The Macmillan Company of Canada Limited, Toronto, 1960. $10.75.

Genetic Perspectives in Disease Resistance and Susceptibility. Annals of the New York Academy of Sciences, Vol. 91, Art. 3, pages 595-818. Conference Editor and Organizing Chairman: Richard H. Osborne. Illust. New York Academy of Sciences, New York, June 7, 1961.

Automatic Process Monitoring. Annals of the New York Academy of Sciences. Vol. 91, Art. 4, pages 819-935. Conference Editor: Sidney Siggia. Illust. New York Academy of Sciences, New York, June 2, 1961.

Pavlovian Conference on Higher Nervous Activity. Annals of the New York Academy of Sciences. Vol. 92, Art. 3, pages 813-1198. Conference Editor: Nathan S. Kline. Illust. New York Academy of Sciences, New York, July 28, 1961.

(Continued on page 610)

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(Continued from page 609)

Handbuch der Orthopädie. In vier Bänden. Band IV. Teil 2. Untere Extremität (Handbook of Orthopedics. In four volumes. Vol. IV. Part 2. Lower Extremities). Edited by G. Hohmann, M. Hackenbroch and K. Lindemann. 1390 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $32.25.

Handbuch der Orthopädie. In vier Bänden. Band III. Obere Extremität (Handbook of Orthopedics. In four volumes. Vol. III. Upper Extremities). Edited by G. Hohmann, M. Hackenbroch and K. Lindemann. 659 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $30.50.

Handbuch der Orthopädie. In vier Bänden. Band IV. Teil 1. Untere Extremität (Handbook of Orthopedics. In four volumes. Vol. IV. Part 1. Lower Extremities). Edited by G. Hohmann, M. Hackenbroch and K. Lindemann. 740 pp. Illust. Georg Thieme Verlag, Stuttgart, W. Germany; Intercontinental Medical Book Corporation, New York, 1961. $33.80.

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